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NONUNION IN SHAFT FRACTURES OF THE HUMERUS*

With Particular Emphasis on the Preoperative Care; and with a Report Of Treatment by Use of the Massive Onlay Graft With Metal Screw Fixation

BY G. E. HAGGART, M.D.,† AND MATTHEW PEELEN, M.D.†

THE relative frequency of nonunion in fractures of the humerus particularly in transverse shaft fractures, has been repeatedly emphasized by various authors. It is also well known that these cases present a difficult therapeutic problem, for not only may the immediate results be failure of bone union, but as Owen points out, nonreunion may recur six to ten months after operation in a humerus which previously appeared to have united, both on clinical and by x-ray examination. Hence, the end result in nonunion of fractures of the humerus should be determined after one year has elapsed from the time of operation.

It is not our purpose to discuss the causes of nonunion as the subject has been exhaustively covered in the literature, but it is worth while to emphasize two factors which appear to us as significant, namely, early operation, that is, at the time of or shortly after fracture occurs, and secondly, inadequate fracture immobilization.

The above statements are based on a review of six patients admitted to the Clinic with nonunion of the humerus in three, of whom bony union was obtained with one operation and in one patient (Case 5) following a second operation. Two patients refused treatment. In two of these cases, the inlay graft was successfully used. In Case 5, the inlay graft was partly absorbed and fractured in the sixth month following operation. At the second operation on this patient, a massive onlay graft with metal screw fixation was employed for the first time with an excellent end result. There was pronounced callus formation in the fourth month. In Case 6, a similar technique also resulted in prompt healing of the fracture. These two cases are reported in detail to illustrate the method now utilized in the Clinic for the treatment of nonunion in shaft fractures of the humerus. In all of the above group, the fractures were located in the mid-third or junction of the middle and lower third of the shaft of the humerus.

In considering the problem presented by nonunion in shaft fractures of the humerus, it is the object of this paper to stress particularly the advantage of adequate preoperative care, which, if followed, we believe materially increases the probability of obtaining bone union; secondly, to emphasize that the massive onlay graft is, in our opinion, the most effective means of obtaining bone union in these cases; and finally, that the operative technique of applying this graft is greatly simplified by the use of metal screws to fix the graft to the humerus. Metal fixation of bone grafts for nonunion has been reported by Hey Groves who employed steel bolts as well as an ordinary split cotter pin (1/16 to 1/8 inch). William L. Estes, Jr. successfully makes use of a special round metal peg with a flange-like head. Metal screws were first employed by us in the treatment of Case 5 (see case report) as a modification of the onlay graft technique. In the end-result studies by Rogers there is reported one case of nonunion of the humerus successfully treated by an onlay graft fixed with metal screws.

PREOPERATIVE CARE

Following nonunion of the humerus, roentgenograms consistently reveal generalized bone atrophy while the tapered ends of the fragments exhibit a pronounced sclerosis to ivory-like bone (figure 6). The extremity, particularly the upper arm, is markedly wasted from disuse due to the unstable humerus (figure 7). Visual evidence of circulatory insufficiency is commonly noted. If infection has previously occurred, there will be additional scarring and fixation of the soft parts. It is obvious that if the above conditions can be improved, even to a relatively slight degree, the chances of bone union are measurably increased. Actually by the plan noted below all of these conditions are decidedly bettered.

The simplest and most effective method to secure this improvement is to permit relatively normal active function of the extremity by means of a stabilizing splint (figure 8). This

*From the Bone and Joint Service of the Lahey Clinic.

†Haggart, G. E.—Orthopedic Surgeon, Lahey Clinic. Peelen, Matthew—Orthopedic Surgeon, Lahey Clinic, 1933-34. For records and addresses of authors see "This Week's Issue," page 849.

consists of a long, laced leather cuff applied to the arm from the upper deltoid region to just above the elbow and connected by a hinge with a similar cuff on the forearm. If there is a wrist drop from radial nerve injury, a light aluminum cockup splint to the hand is provided, attached to the forearm cuff. The patient is then instructed to use the arm in as nearly normal a manner as possible. The actual degree of function obtained by the use of this apparatus is quite striking as compared with the former marked limitation of arm activity. The previously wasted muscles regain power, circulation is greatly improved and by x-ray there is a progressive decrease of the bone atrophy. The length of time that the splint is worn is determined by the original condition, by the rapidity of improvement in the functional appearance and by the x-ray evidence of diminishing bone atrophy. The average duration in these cases was five months.

We wish to emphasize, however, that in the above patients nonunion had been present from one and one-half to three years and that these individuals had all previously submitted to surgery on the humerus. One patient had three operations, another two, and two patients had one unsuccessful operation to secure union of the fracture. In each instance our roentgenograms revealed pronounced bone atrophy of the fragments. There was evident circulatory disturbance in the extremity as well as advanced muscle wasting. Slight to moderate loss of radial nerve cutaneous sensation was noted in three patients, associated with weakness in the extensor muscles of the forearm.

This particular group of patients would obviously require a relatively long interval of preoperative care. In cases seen earlier, however, without previous operation, and in whom bone atrophy, muscle weakness and local circulatory insufficiency were not so marked, it was evident that the preoperative treatment would be of shorter duration than is here reported.

BLOOD CHEMISTRY

In all patients exhaustive blood chemistry studies were done with repeated estimations (in operated cases) of the blood calcium, phosphorus, and phosphatase. With one exception the findings were normal. The "product" of calcium phosphorus determinations was not significant. In one patient, Case 5, the blood phosphorus averaged 1 to 1.2 milligrams on four examinations. This value is lower than the average blood phosphorus estimation (2.5 to 4 mgm. per 100 cc. blood). Therefore phosphorus, in the form of dibasic calcium phosphate, was administered by mouth for five months, when the blood phosphorus averaged 3 to 4.4 milligrams per 100 cc. of blood.

DIET

All patients were given a high vitamin, high protein diet with adequate calcium and phosphorus bearing food. The obese patients were reduced, and cases of malnutrition advised a high caloric intake. The bowel function was supervised and in fact every effort made to improve the patient's physical and mental status. In each instance the individual's general condition at the time of operation was decidedly better than when first seen.

THE ONLAY GRAFT WITH METAL SCREW FIXATION

Sever sums up the requirements for treatment of nonunion in fractures of the humerus when he states that the operative repair must provide primary internal, as well as secondary external fixation for a long period. This author also remarks that external fixation is never complete in any apparatus.

We feel that primary *internal fixation* by autogenous bone graft is especially important. In our hands, such fixation is best secured by the use of the massive onlay graft held by metal screws. While two of the cases in this series were successfully treated by the inlay graft technique, the degree of primary internal fixation obtained by us through this method, does not compare with the firm fixation secured through use of the onlay graft. Furthermore, in patients with nonunion of the humerus a "carpenter's fit" for the inlay graft is difficult to obtain in the slender, atrophied fragments and yet not injure the adjacent thin margins of humeral bone.

The reader is referred to the articles by Mitchell, Henderson and by Campbell for a detailed description of the general technique of application of the massive onlay graft. The significant modification in our hands is the use of metal screws to fix the graft.

The technic of looped chromic catgut sutures (Mitchell) to hold the graft in place has not, in our opinion, provided sufficiently firm internal fixation for nonunion of the humerus. In the sclerotic and atrophic humerus we have found that beef bone (Henderson) or autogenous screws or nails (Campbell) do not hold so firmly as a self-tapping metal machine screw (Sherman). Furthermore, bone screws are rather friable and may break off when partly inserted. Finally, we believe that the use of taps and dies to make autogenous bone screws while ideal is not satisfactory because of the time consumed and because of the fragility of normal human bone.

The advantages, therefore, of metal screw fixation of the massive onlay graft may be briefly summarized as (1) the simplicity and ease of application of the graft as compared with the use of beef bone screws or autogenous bone nails

or screws, (2) the very firm internal fixation secured, resulting in close contact between graft and fragments, and (3) with the use of metal screws a corps of assistants is not necessary.

The presumable disadvantage of metal screws is that as a foreign body they might prevent bone union, or later becoming loose have to be removed. But in the two cases so treated there was no evidence of delay in bone union as the x-rays revealed advanced callus formation within three months' time (figure 3c). Over an interval of two years and sixteen months respectively, the roentgenograms do not exhibit any signs of bone reaction or absorption about the metal screws (figures 4 and 10).

Metal screws and nails have been, and are of course now, repeatedly utilized by surgeons for fixation of loose bone fragments. In the absence of bone reaction there is a difference of opinion as to whether the metal should be removed. Ashurst, for example, reports patients in whom metal screws or nails remained twenty years without ill-effect. In the cases here reported removal of the metal screws is a simple procedure. All patients are warned that this may be necessary but only in the event of local symptoms or of x-ray signs of bone reaction about the screws. We conclude, therefore, that the available evidence does not contraindicate the use of metal screws to hold an onlay graft applied for nonunion of the humerus.

ACCESSORY CHIP AND SPLINTER GRAFTS

In addition to the massive onlay graft it is also most important to surround the fracture site with splinter and chip grafts of cortical and particularly of cancellous bone (figures 3b and 11) likewise removed from the tibia, preferably from near the epiphyseal line. The square surface of raw bone is thereby greatly increased and in addition a large local supply of calcium is available so that the probability of bony union is still further assured.

NONUNION WITH INFECTION

In patients with nonunion of the humerus following fracture and associated with infection the bone graft operation as herein described is not undertaken until one year after the closure of all wounds, particularly in the presence of considerable scar formation. When the time for operation is determined the procedure is divided into two stages. The scar is first excised and if this operation is not too extensive the bone fragments are also prepared for the graft and the wound then closed. If the immediate postoperative course is uneventful the wound is then reopened in fourteen to twenty-one days and the onlay graft applied.

In this type of case, that is, where there has been infection, Phemister reports that if the infection can be sufficiently well localized, and hence probably avoided at operation, much time and disability may be saved by the use of the onlay splint graft. The latter is not fastened to the humerus but simply placed on the two fragments, with the fracture area as the midpoint. To succeed in such an operative attack, in the absence of any internal fixation, it is additionally required that the bone fragments be held in relatively good alignment and position by preëxistent callus or scar.

OTHER METHODS

The following brief statements summarize our views on other commonly employed methods of bone grafting.

The intramedullary peg graft is decidedly not satisfactory for treatment of nonunion of the humerus.

The osteoperiosteal graft alone is not adequate chiefly because of lack of internal fixation. This we believe to be true even if step cutting and suturing of fragments are performed. Such a graft may often be advantageously employed as an accessory aid to bone union; or, in those instances where immediate fixation by the graft is not necessary, that is, when the alignment and approximation of fragments is acceptable and fixation is secured by a combination of preëxisting callus plus the externally applied apparatus.

The sliding bone graft appears preferable to the above two methods, but because of the atrophic and often sclerotic bone necessarily utilized, we do not believe that this procedure is as effective as a massive onlay graft from the tibia.

We have had no experience with ivory plates or pegs.

Steel plating in these cases is unwise, while silver wire is to be condemned.

COMMENTS ON THE OPERATIVE TECHNIQUE OF ONLAY GRAFT WITH METAL SCREW FIXATION

It is decidedly advantageous to apply a snug body cast with shoulder straps two days before operation. Following application of the bone graft, a close fitting arm plaster, extending from the proximal hand to shoulder, is then attached to the body cast. This cast is worn until roentgenograms exhibit bone union—an average of three to five months. If the cast is applied in the manner described, it is possible to obtain the maximum degree of firm, postoperative external fixation. Such immobilization is essential to secure healing of the fracture.

The skin incision should be ample so that there is no trauma to the soft parts from retraction. The exposure of the humeral shaft (figure 12) as described by Henry affords easy access to the bone with minimal bleeding and complete protection of the radial nerve.

The periosteum of the humerus is incised and

gently separated over an area sufficient only to admit placing the graft directly on the humerus. In this connection we have not found clinical evidence that the periosteum alone is of osteogenetic significance. However, the periosteum is vitally important in maintaining bone circulation as emphasized by Gallie and Robertson.

As Henderson suggests, the eburnated ends of the bone fragments are easily prepared by a transverse saw cut, thus removing the greater part of the sclerotic bone. Drilling (with a 1/4 inch bit) of the medullary canal may also be done.

The bone bed in the fragments upon which the graft is to lie is most simply fashioned by using a broad osteotome and removing a thin layer of cortex down to bleeding bone (figure 13).

The average size of the onlay graft in these cases is one-half inch wide, six inches long and in thickness the depth of the tibial cortex down to the medullary canal. The necessity of a large graft cannot be too strongly emphasized. The graft is easily removed from the tibia by motor saw and chisel.

The bone fragments then are aligned and the graft held in place by an assistant. Holes are drilled through the graft and the superior cortex of the humerus with a special bit measuring one thirty-second of an inch less in diameter than the diameter of the metal screws (9/64 of an inch). The latter are the self-tapping vanadium machine screws devised by Sherman. When finally in place, the screws pass through the graft and *both* cortices of the humerus (figure 13). The resulting fixation is so firm that the extremity feels like the normal arm.

After placing multiple bone chips and splinter grafts about the fracture, the wound is closed in layers and the plaster spica completed.

CONCLUSIONS

1. Nonunion in shaft fracture of the humerus most frequently occurs in the mid-third and junction of the mid and distal third of the humeral shaft.

2. Of the various factors which may cause nonunion we are impressed by (1) early operation, that is, performed at the time of or shortly after the fracture and (2) faulty fracture immobilization.

3. To obtain bone union the importance of adequate preoperative care is emphasized. Such a régime is directed toward improving arm circulation, to overcoming muscle weakness and to diminishing the atrophy of the bone fragments. This is accomplished by instructing the patient to wear an arm splint designed to immobilize the unstable fragments of the humerus and so permit relatively normal arm function. As explained in the text the length of time the splint is worn is an individual problem. With nonunion of long duration and the consequent marked atrophy of bone and soft parts, as in the patients here reported, the preoperative care averaged five months. In addition every effort is made to improve the patient's general physical and mental status through proper diet, rest and good hygiene.

4. In our opinion the most satisfactory operative procedure in these cases is that of the massive onlay graft with metal screw fixation. The use of metal screws greatly simplifies this operation. The case histories of two patients treated by this method are reported in detail. In both instances a good result was obtained.

CASE 1. Mrs. N. B., housewife, aged twenty-nine years, was admitted to the Clinic on August 30, 1932 for treatment of nonunion of a fracture of the left humerus.

In April, 1931 the patient was in an automobile accident and sustained a simple comminuted and spiral type of fracture of the shaft of the humerus at the junction of the middle and lower third. Immediate wrist drop and loss of sensation over the dorsum of the hand and forearm were reported. This condition gradually improved by slow progressive stages so that on admission to the Clinic there remained superficial anesthesia of the dorsal forefinger, together with moderate weakness of the extensor muscles, thumb and distal forearm. Within three hours after the injury open reduction was performed elsewhere. Apparently the fragments were sutured with catgut. The arm was then held in a metal splint for four weeks. One year after the injury, also elsewhere, an intramedullary bone peg graft was inserted between the humeral fragments. The patient was immobilized six weeks. Nonunion persisted.

On admission to the Clinic there was obvious complete instability of the humerus (figure 1).

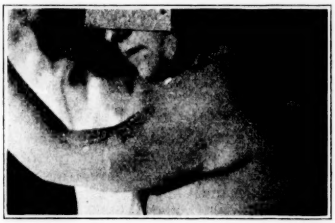


FIGURE 1: Case 1—Admission photograph. Note scar of previous operations and that to raise arm it must be supported.

Range of wrist motion was normal, but there was definite weakness in the grasp of the hand.

The x-rays (figure 2) revealed marked generalized atrophy of the bone of the humerus and sclerosis of the ends of the fragments. No attempt at bone repair appeared. The only laboratory note of significance was a blood phosphorus of 1.8 milligrams. All other findings were normal. The general health of the patient was excellent, although she was somewhat overweight.

a

b

FIGURE 2: Case 1—a, b—Admission x-rays. Generalized bone atrophy is evident, and there is sclerosis of the ends of the fragments with no evidence of repair.

Preoperative Treatment: Carried out as described above. Dibasic calcium phosphate by mouth raised the blood phosphorus at the end of four months to an average level of 3 to 3.4 milligrams. A leather arm splint permitted active function of the extremity, so that at the end of five months there was marked improvement in the circulation and muscle development in the arm, while bone atrophy was appreciably less.

Operation: On January 23, 1933 an inlay graft of bone removed from the tibia was done; splinter and chip grafts placed about the fracture line and the arm then immobilized in a plaster spica. May 8, 1933. Five months postoperative; roentgenograms showed maintenance of alignment of the fragments and apparent early healing of the fracture. On July 28, 1933 further x-rays revealed fracture of the bone graft. The position of the humeral fragments was unchanged. Plaster cast support was therefore continued. But on October 25, 1933, ten months following the operation, while the graft had united to the proximal fragment of humerus there was definite absorption of the graft at the site of fracture and complete failure of bone union of the humerus.

February 10, 1934, a second operation consisting of application of a massive onlay graft with metal screw fixation was done. Multiple chip and splinter grafts were also placed about the site of the fracture (figure 3). The arm was immobilized in a plaster spica for five months. Union was obviously underway in the fourth month, and five months following the operation the fracture had completely healed. The patient regained normal function of the extremity within one month from this time and since then has continued to use the arm in a normal manner.

Figure 3: Case 1-a

b

c

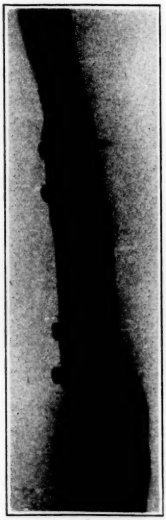
FIGURE 3: Case 1—a—X-rays taken just before the second operation, consisting of the application of a massive onlay graft. Note slight lateral bowing of proximal fragment of humerus due to angle of union of part of inlay graft.

To prevent excessive shortening of the humerus it was decided preoperatively to accept this deformity. It was thought that with bone union obtained continued bone growth would tend to correct this angulation progressively (compare figure 5).

b—Immediate postoperative A-P x-ray showing massive onlay graft in place, and fracture line surrounded by multiple chip and splinter grafts.

c—Lateral roentgenogram, three months postoperative. (The immediate postoperative lateral film was not sufficiently clear to reproduce.) Note extensive callus and beginning union.

a



b

FIGURE 4: a, b Case 1—Two year follow-up. Firm bone union. There is no roentgenographic evidence of reaction about the metal screws.

February 12, 1936—Check-up roentgenograms, taken two years following the operation (figure 4), do not show any reaction about the screws and reveal firm union of the fracture. The patient reports no symptoms. Function of the arm is normal (figure 5).

a

b

FIGURE 5: a, b Case 1—Two year follow-up. No symptoms. Normal function of the arm. Note comparison of arms reveals no deformity.

Summary of Case 1: Ununited fracture of three years' duration in a twenty-nine year old housewife and located at the junction of the middle and lower thirds of the shaft of the left humerus. Two previous operations before admission to the Clinic. An additional attempt by us to obtain union with an inlay graft was unsuccessful. This graft fractured during the sixth postoperative month and was in greater part absorbed by the tenth month after the operation. The patient was re-operated on February 10, 1934 with application of massive onlay graft held by metal screw fixation, together with multiple chip and splinter grafts placed about fracture line. Union was obtained.

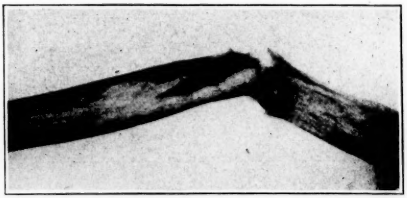
Follow-Up: Two years after the operation there is normal function of the arm, while the roentgenograms reveal no evidence of reaction about the metal screws. The patient carries on her usual activities.

CASE 2. Mr. A. H., student, aged twenty years, was admitted to the Clinic March 24, 1934 for treatment of nonunion of fracture of the middle third of the shaft of the right humerus.

The patient fell from a ladder and fractured the humerus one and one-half years before admission. A closed reduction was twice attempted elsewhere but the fracture did not heal. Six months later, also elsewhere, a bone graft from the tibia was applied (the exact type of procedure not known) but nonunion persisted.

Examination revealed an ununited fracture at the middle third of the shaft of the right humerus with complete flail motion. The x-rays (figure 6) exhibit

a



b

FIGURE 6: a, b Case 2—Admission roentgenograms; characteristic pseudoarthrosis with generalized bone atrophy of the humerus.

FIGURE 7: Case 2—Admission photograph. Observe the pronounced muscle atrophy.

FIGURE 8: Case 2—Illustrating the type of brace worn to encourage active function of the extremity in order to improve the arm circulation, develop musculature and overcome bone atrophy of the humerus.

pronounced generalized atrophy of the fragments of the humerus and the usual sclerosis at the end of the fragments. The third fragment was considered to be probably part of a bone graft from the previous operation. As seen in figure 7 there was marked muscle atrophy of the extremity, the circulation was inadequate and disability complete. General physical examination and a detailed blood study was in no sense remarkable except that the patient was rather slender and apparently somewhat underweight.

a

b

FIGURE 9: a, b—Case 2—Sixteen months follow-up. Normal arm function, no symptoms. Compare with figure 7.

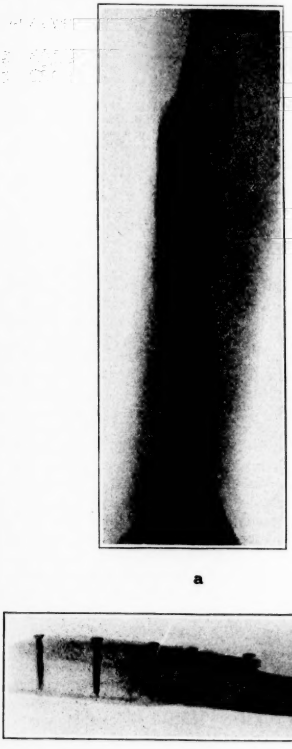
Preoperative Treatment: The patient was placed on the régime as described. He wore the supporting arm brace for six months (figure 8) at the end of which time his general condition was considerably improved. The musculature of the arm was then decidedly stronger while the circulation was comparable to that of the uninjured arm. In addition the roentgenograms revealed a generalized increase in density throughout the shaft of the humerus.

On October 19, 1934 an onlay graft with metal

screw fixation was applied together with multiple chip and splinter grafts about the site of the fracture. The extremity was then immobilized in a plaster spica.

Postoperative Course: In the third month the roentgenograms showed evidence of beginning callus formation which was sufficiently advanced in the fourth month, so that the arm was removed from the plaster spica. On clinical and x-ray examination there was firm union of the fracture. The patient rapidly regained normal function of the extremity (figure 9).

Follow-Up: February 19, 1936, sixteen months after operation; x-rays (figure 10) show a healed fracture and no evidence of any reaction about the metal screws. The patient has normal function of the extremity and has resumed his usual activities.



a

b

FIGURE 10: a, b—Case 2—Sixteen months follow-up. Roentgenograms show solid union and no evidence of reaction about the metal screws.

Summary of Case 2: Ununited fracture mid-third shaft of right humerus of one and one-half years' duration in a twenty-year old student. One pre-

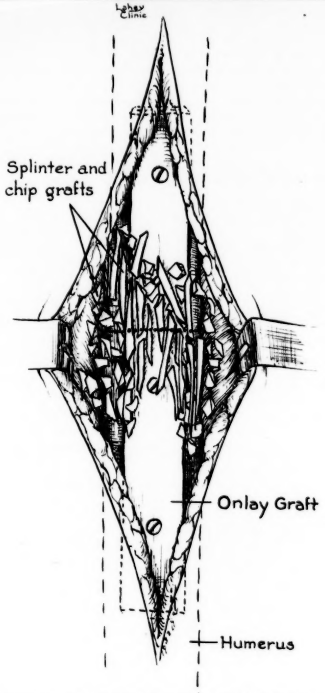


FIGURE 11: To illustrate placing of multiple bone chip and splinter grafts about the fracture line following the application of the onlay graft. The heavy dotted line represents the cut surfaces of the humeral fragments fitted together.

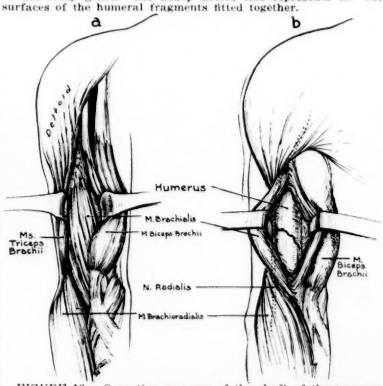


FIGURE 12: Operative exposure of the shaft of the humerus through an essentially avascular field. By splitting the fibers of the brachialis muscle the bone is exposed and at the same time the radial nerve is protected.

vious operation to obtain union one year before admission. The patient is of frail physique with marked bone and muscle atrophy of the injured arm. He was placed on the preoperative régime described for six months, with marked local and systemic improvement.

Operation October 19, 1934 consisted of a massive onlay graft held by metal screws with multiple chip and splinter grafts about the fracture line. Immo-

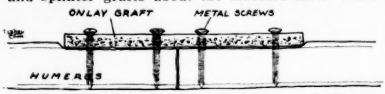


FIGURE 13: Line drawing to illustrate; bed of bleeding bone in the fragments upon which the graft is placed; the contact of the cut ends of the fragments; and the firm fixation obtained by use of metal screws which pass through the graft and both cortices of humerus.

bilized four months with firm union. Sixteen months follow-up shows a good anatomic and functional result. The roentgenograms do not exhibit evidence of reaction about the metal screws.

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THE HEART IN CHRONIC GLOMERULAR NEPHRITIS*

BY ARTHUR B. RICHTER, M.D.,† AND JAMES P. O'HARE, M.D.†

WHILE most of our profession now appreciate the importance of the extrarenal elements in one form of Bright's Disease—chronic vascular nephritis—there are many who still regard glomerular nephritis solely as a disease of the kidneys. The first in a series of papers on this form of nephritis[1] pointed out that the eyeground changes found in glomerular nephritis are vascular in origin and that they are essentially the same lesions as those found in nonrenal vascular hypertension. Another paper, to be published later, will show that a significant number of patients with this type of nephritis die from causes other than their renal insufficiency. This paper is a study of the part played by the heart in chronic glomerular nephritis.

While the literature fairly teems with reports on the hypertensive heart there is a great paucity of writing on the cardiac changes in glomerular nephritis *per se*. To be sure, all textbooks refer to its hypertension and cardiac hypertrophy and the ominous significance of uremic pericarditis. Special articles are, however, few indeed and concerned chiefly with special phases of the subject. Barach[2] and Pyrah[3] write specifically of pericarditis. Wood and White[4] and Schwab and Herrmann[5] in their respective articles disagree on the interpretation of the electrocardiograph changes found in uremia.

Such a scarcity of reports is easy to understand. In the first place, in acute nephritis the heart is a matter of concern only in the very rare patient in whom hypertensive crises or convulsive seizures bring about congestive failure. Then again in the entire group of patients with cardiac involvement the number of cases of chronic glomerular nephritis is, after all, relatively small. Finally most authors assume—perhaps correctly—that the heart in chronic glomerular nephritis is merely the ordinary hypertensive heart. Our chief purpose in making this report is the twofold one of remedying this lack of established data in the literature and of adding to the existing knowledge of pericarditis in nephritis.

A Renal Clinic in existence for eighteen years has given us an unusual opportunity to follow a fairly large group of these patients throughout a large part of their illness. Our report is based on a study of all patients with significant chronic glomerular nephritis who have come to autopsy at the Peter Bent Brigham Hospital from its opening in 1913 to the beginning of the year 1935. From a total of seventy-five such cases, five have been excluded for complicating cardiac valvular disease and four because they were examples of that unusual and complicated syndrome described by Libman and Sachs[6], Tremaine[7] and Christian[8] of long-continued fever with inflammatory changes in serous and synovial membranes, subsequent glomerular nephritis and chronic fibrous pericarditis.

*From the Medical Clinic of the Peter Bent Brigham Hospital, Boston.

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This is the third in a series of papers on Chronic Glomerular Nephritis.

†Richter, Arthur B.—Assistant Resident Physician, Peter Bent Brigham Hospital, 1935. O'Hare, James P.—Senior Associate in Medicine, Peter Bent Brigham Hospital. For records and addresses of authors see "This Week's Issue," page 819.

Of the remaining sixty-six patients, forty-one were males and twenty-five females, varying in age when first seen, from 2.6* to sixty-two years. One-third of our sixty-six patients were observed periodically from one to ten years. Another third were followed from one to twelve months. Observation in the remainder was limited to a brief but variable period antedating death.

It is obvious that in nephritis as in any other chronic disease death may occur not from the primary disease but from related or wholly unrelated causes. This was so in seven of our patients who died not from uremia but from such extrarenal causes as cardiac failure, cerebral hemorrhage, pneumonia, etc., with renal function still adequate. In these seven the final blood urea nitrogens averaged 37 mgm. per cent. In the remaining fifty-nine patients, death occurred in uremia with renal insufficiency practically complete. In them the blood urea nitrogens reached a terminal level averaging 172 mgm. per cent.

In the entire group of sixty-six, hypertension of moderate or severe grade (average 200/120) was present in all but five. The low pressures in the latter number are easily accounted for by the fact that they were seen only a few days before death and no doubt their pressures represented merely the prelethal fall.

SYMPTOMS

It is well known that most patients with a dry chronic glomerular nephritis have few or no complaints until the process is far advanced. This is particularly true of the cardiac phase of the disease. Occasionally one does see a patient in whom cardiovascular lesions outstrip the renal disease. In such a patient cardiac problems may appear early and play a predominant rôle.

While cardiac disturbances of one kind or another developed in every one of our patients, the time of onset, the type and the severity varied considerably. Complaints about the heart were rarely encountered until one year before death, with the exception of certain significant differences that did occasionally occur. These will be discussed later. The cardiac symptoms and signs differ but little from those seen in primary hypertension.

Easy fatigue and flatulence, possibly of cardiac origin, occurred frequently and early but these symptoms as well as the palpitation and vague apical discomfort, which were less often and more tardily noted, were difficult to evaluate. The two outstanding symptoms were pain and dyspnea and these rarely occurred until one year before death.

Cardiac pain apart from vague and insignificant apical distress was uncommon and when present was due either to coronary artery disease or to pericarditis. Angina pectoris was a symptom in only one patient and typical coronary thrombosis likewise occurred in but a single patient, a lad of twenty-eight. This is a much lower incidence than in primary hypertension in which the age of the patient is greater and the arterial disease is usually of longer duration and of greater severity.

Uremic pericarditis, occurring in 44 per cent of our sixty-six patients, was a more common cause of precordial discomfort. In sixteen patients it took the form of precordial distress or oppression but in five it was described as severe pain. In eight patients in whom a rub was heard the patient was unaware of any cardiac difficulty. In two cases the precordial pain, associated with slight fever, leucocytosis, a fall in blood pressure, a friction rub and even electrocardiographic changes so closely simulated coronary artery thrombosis that the true diagnosis became apparent only after the discovery of the urinary abnormalities and the high blood urea nitrogen.

Dyspnea was by far the most prominent cardiac symptom occurring in thirty-two cases. As a rule, patients first began to complain of this on exertion about one year—occasionally longer—before death. As the disease progressed with an increase in the diastolic pressure and gradually developing cardiac and renal failure, shortness of breath became more severe. Shortly before death it was present in practically every patient.

In the late stages the dyspnea was not always exclusively cardiac. Frequently cardiac failure was complicated by severe anemia, acidosis and a failing cerebral circulation making it very difficult at times to evaluate satisfactorily the various components that brought about the difficult breathing. Typical Kussmaul breathing was occasionally observed, but Cheyne-Stokes dyspnea was much more frequent. Still another form of difficult breathing, recurring paroxysmal dyspnea or acute pulmonary edema, was an important feature in six cases. In one of these the old-fashioned sweat bath was responsible for a fatal attack. Another patient was of especial interest because his dyspnea was of such striking severity that some unusual pulmonary complication in addition to cardiac failure seemed evident. Autopsy revealed multiple thromboses of the smaller pulmonary arteries and numerous pulmonary infarctions.

SIGNS

While cardiac symptoms are relatively few in glomerular nephritis and important only late in the disease, cardiac signs, as one would expect in hypertensive patients, are common, and may occur early with the development of the permanently elevated blood pressure. Their significance varies with the individual signs.

*Three cases were first seen at the Children's Hospital of Boston. We are indebted to this hospital for the early data.

SIZE OF THE HEART

An accurate determination of the duration and the degree of cardiac hypertrophy is one of the greatest aids in evaluating the cardiac status of the hypertensive patient. The hypertrophied and dilated heart is destined to failure. No accurate method for determining these factors is available and we are compelled to rely on the somewhat uncertain clinical determination of the position and force of the apex impulse and percussion measurements together with roentgen study.

The location of the apex impulse, together with the percussion measurements, was repeatedly recorded in the forty-four cases that were followed for a considerable period before death. By these clinical methods alone the heart seemed to be of normal size in only eight. It was slightly to moderately enlarged in fifteen, and moderately to markedly enlarged in twenty-one cases. A rapid increase in size with onset of cardiac failure was noted in an occasional patient. In most instances the greatest degree of cardiac enlargement was found in those cases in which severe hypertension had been present for over a year.

Repeated x-ray studies, consisting of a fluoroscopic examination and a seven-foot film, were available in eighteen patients. By methods of physical examination alone in these cases the heart was moderately to markedly enlarged in twelve, slightly enlarged in two and apparently of normal size in four patients. In this same group by teleroentgenograms twelve of the eighteen cases had definitely enlarged hearts, five of severe grade. Of the six cases with a normal cardiothoracic ratio, roentgenoscopy showed evidence of hypertrophy of the left ventricle in two. The other four with hearts of normal size by Roentgen ray were also normal on physical examination.

In six cases serial teleroentgenograms over one to five years showed progressive cardiac enlargement, a feature not detected with reliable accuracy by physical examination. In three of these a rapid increase in size accompanied cardiac dilatation, the diagnosis being confirmed at the same time by a feeble beat on fluoroscopy. In another the heart was of normal size until an attack of lobar pneumonia. During the following ten months the heart enlarged so rapidly that acute myocarditis was suspected. However, the fluoroscope revealed no weakening of the heart beat and necropsy revealed merely a markedly hypertrophied heart.

On roentgenologic examination the aorta was tortuous in over half of the eighteen patients examined. Most of these were in the older age groups. In none was there dilatation above the accepted normal upper limit of 6 cm. in the left oblique view.

HEART SOUNDS

Even when the heart sounds were carefully described in patients followed for several years, the quality, intensity, and splitting of sounds seemed to have but little significance except, perhaps, at the very end. In forty-one out of fifty-six patients the second sound at the aortic area was of increased intensity, accompanied in most instances by an accentuated first sound at the mitral area. This is to be expected with the elevated blood pressure. Diminished intensity of the first sound at the mitral area was only rarely noted.

ARRHYTHMIAS

Prior to the onset of uremic pericarditis clinical irregularities of the heart beat were uncommon. Of the sixty-six cases, premature beats in six and auricular fibrillation in one were the only arrhythmias noted. Those occurring after pericarditis developed will be discussed later.

GALLOP RHYTHM

A gallop rhythm, usually protodiastolic in type, was recorded late in the disease in one third of the sixty-six cases; twelve of these patients had congestive failure. Most of these patients with gallop rhythm died within six months. In this connection, however, it should be remembered that the element of progressive renal insufficiency is of more importance than the cardiac one in the majority of these cases of chronic glomerular nephritis. Therefore, this prognostic significance of gallop rhythm can hardly be applied to primary hypertensive patients without renal failure.

MURMURS

In the entire group of sixty-six patients twenty-three showed no murmurs of any kind during the entire period of observation, for some as much as ten years. A fair number of these were, too, severely hypertensive. In forty-three a systolic or diastolic murmur was found at some time during the period the patients were followed. Thirty-seven had a precordial systolic murmur of greatest intensity in the mitral area, these being described as "faint" in seventeen cases, moderately "loud" in twelve, and "loud" in eight cases. A systolic murmur of moderate intensity was sharply localized to the mitral or aortic areas in three cases each. Three more had transient apical systolic murmurs. In ten patients the systolic murmur appeared only with the development of hypertension.

An early blowing diastolic murmur at the aortic area, along the left sternal border, and rarely at the mitral area is not very rare in hypertension. In such cases the heart valves and rings are usually normal at necropsy. A

diastolic murmur of this sort unaccompanied by a thrill, was present in ten cases. In at least three instances it appeared after the onset of hypertension. In two cases the murmur was transient and present only during periods of extreme elevation of the blood pressure. Although anemia was present in the late stages of the nephritis in all of the cases it could not be considered even a possible factor in the production of the diastolic murmurs except in two patients both of whom were adolescents. In one of the six cases in which the diastolic murmur was most typical of aortic insufficiency true structural disease of the aortic valves was suspected. At necropsy, however, a markedly dilated pulmonic valve ring was the only valvular abnormality. Of the entire series this case was the only one with appreciable dilatation of a valve ring and in none of the cases did the leaflets show evidence of endocarditis. In four of the ten cases the diastolic murmur, occurring early in diastole, was most distinct at the mitral area, resulting in a questionable diagnosis of mitral stenosis in each instance. The presence of diastolic murmurs did not appear to influence the course of the disease in these ten patients, nor was it an apparent factor in the congestive failure which was present in a third of them.

CONGESTIVE HEART FAILURE

Before the terminal stage of chronic glomerular nephritis, congestive failure becomes the most important clinical feature in about one fourth of the cases, as contrasted with primary hypertension where the cardiac factor is the significant one in about two-thirds of the cases. Probably this is due to the greater duration and degree of hypertension in the latter group, and to the fact that only about 10 per cent of the patients with primary hypertension die of renal failure. In the primary vascular group the average age of the patients is greater, the incidence of coronary artery disease higher and the degree of cardiac hypertrophy usually greater.

In the present series congestive failure became an important clinical feature and a few months before death in 23 per cent. Left-sided failure characterized by paroxysmal dyspnea was the outstanding feature in five patients. As mentioned above, one died in an attack of pulmonary edema produced by the use of the sweat bath. Eight of the ten cases with congestive failure had the usual signs of a failing right heart, i.e. orthopnea, severe ankle edema, engorged liver, râles, hydrothorax, etc. In the other two cases the principal feature of the right heart failure was recurrent ascites, which led to a consideration of cirrhosis of the liver. Abdominal pain due to an engorged liver was so severe in another patient that an acute surgical condition in the abdomen was at first suspected.

In the terminal stage of the nephritis, edema is usually present and is nearly always cardiac in origin and distribution. Late in the disease all but five of the sixty-six cases had slight to moderate ankle and sacral edema. Right heart failure with generalized edema in four patients and left failure in one (pulmonary edema) was precipitated by forcing fluids to about 3000 cc. in twenty-four hours. This is an adequate warning to those who would force fluids in uremia without considering the enfeebled circulatory mechanism in the terminal stages of chronic nephritis. Two cases with a nephrotic component had generalized edema believed to be both cardiac and renal in origin, the rapid development of renal failure having been accompanied by hypertension and symptoms of cardiac decompensation.

PERICARDITIS

One of the most productive parts of our study concerned the pericarditis found in the uremic stage*. In the literature the average incidence of acute fibrinous pericarditis in all types of Bright's disease studied clinically and pathologically is approximately 10 per cent. In fifty-nine patients with glomerular nephritis who died in uremia a pericardial rub was heard in 36 per cent. Including those cases in which the acute pericarditis was found only at necropsy we find a total incidence in the fifty-nine cases of glomerular nephritis of 48 per cent. This unusually high incidence can be explained only by the fact that we were especially looking for the rub or because our series of cases, though large for chronic glomerular nephritis, is after all relatively small. However, the former explanation seems more probable in view of the fact that the frequency of acute pericarditis in a parallel group of fifty-five patients who died from vascular nephritis was 24 per cent, more than twice that given in the literature for unclassified nephritis. Our total incidence for both groups of cases was 36 per cent.

In our patients the friction rub varied from the extremely soft and evanescent one to the very coarse one which persisted to death. The former is not infrequently missed because one does not happen to listen at the right time. The coarse rub, in our experience, is more lasting and, therefore, rarely missed. In our entire group the rub disappeared permanently in only three patients.

Uremic pericarditis is one of the most important prognostic signs in all medicine, the importance of this sign lying in the fact that death occurs ordinarily within a few days to a few weeks after it appears. Barach[2], however, has reported three patients who have lived from two to twelve months after a rub was heard. In our entire group of fifty-nine patients the

*The interest and importance of our observations warrant a separate and detailed report which will be published elsewhere. Only a summary of our findings will be given here.

interval between the appearance of the rub and death varied from one to thirty-six days with an average of seven days.

As for signs other than the rub which has been described previously there were but few. Pericardial effusion was commonly present at necropsy but this condition was diagnosed clinically in only one patient on the basis of an increased area of cardiac dullness and diminished intensity of the heart sounds. The pulse and temperature rose moderately in an occasional case, and the blood pressure remained at its previous level except in the rare patient where a fall led to some difficulty in eliminating from consideration coronary thrombosis.

By far the most striking signs were the disturbances of the cardiac mechanism and various electrocardiographic changes.

ELECTROCARDIOGRAPHY

Electrocardiograph studies were available in thirty-eight of our patients. All had hypertension and about half were on a maintenance dose of digitalis. Eight had definite pericarditis. On the whole the electrocardiographic changes occurring prior to the onset of pericarditis seemed to be of minor importance. If we except the eight cases of pericarditis, arrhythmias were noted in but four patients. Three of these showed premature beats and in one there was auricular fibrillation. The thirty nonpericarditic patients may be divided into two groups, those with proved coronary arteriosclerosis and those without such. Of the former there were eleven with moderate to severe coronary artery sclerosis at necropsy. All of these had at least one tracing within two months of death. The electrocardiograms of four were normal; two showed only left axis deviation while in four more there was an additional T wave negativity in all three leads, and a low voltage of the ventricular complex in one case. The electrocardiographic changes of the one patient with coronary thrombosis were those of posterior infarction.

In the remaining nineteen patients without obvious coronary arteriosclerosis at necropsy the electrocardiograms in general differed but little from those in the group of eleven cases with definite coronary artery sclerosis at necropsy. A small proportion, two out of nineteen, showed negative T waves in all three leads. In four, the T waves were inverted only in leads I and II. Five cases with normal tracings developed, over a period of months, left ventricular preponderance or negative T waves, rarely both. In about 10 per cent of the series the so-called high voltage electrocardiogram was obtained[9].

Wood and White[4] in a study of the electrocardiogram in uremia and severe chronic nephritis described positive deviations of the RS-T segment, T wave negativity, and abnormalities of rhythm and conduction and attributed these to the effect of the uremic toxins on the myocardium. Pericarditis was described in at least two of these cases. Levine[10] in 1929 presented a case of uremic pericarditis with positive deviations of the RS-T segment similar to those described by Wood and White. Recently Schwab and Herrmann[5] offered proof that pericarditis was the probable explanation of these T wave changes.

A study of our tracings seems to support Schwab and Herrmann. It is significant that no changes except the ones usually found in hypertensive patients occurred in our cases until pericarditis or myocardial infarction had developed. This, of course, does not absolutely exclude uremia or coronary artery disease as a factor.

Let us see what light our data throw on the possible relationship of uremia to the abnormalities found. Quite arbitrarily we have considered that uremia was present when the blood urea nitrogen was 70 mgm. per 100 cc. or more. For the purpose of comparing the electrocardiographic changes in various periods of the nephritis we divided the patients into three groups as follows:

(1) Sixteen cases with two or more tracings before uremia developed.

(2) Fifteen cases in which one or more tracings were taken during well-marked uremia before the appearance of the pericardial rub.

(3) Eight cases with a total of fourteen electrocardiograms taken while a pericardial rub was present. All of these cases had hypertension. Digitalis effects need not be considered because the drug had been discontinued in all for some time because of vomiting.

Prior to the onset of pericarditis these cases had showed only such minor electrocardiographic changes as axis deviation, T wave negativity, low voltage of the ventricular complex, and an occasional ectopic beat. With the development of pericarditis, all of the eight cases except one displayed abnormal electrocardiograms. A positive deviation of the RS-T segment was present in leads I and II in one patient and in all leads of another. The T waves were upright resembling those described by Wood and White. In both patients normal tracings had been obtained a few days prior to the onset of pericarditis. In one of them there was not merely deviation of the RS-T segment but also transient auricular fibrillation and premature nodal beats. Chest leads were normal suggesting that the coronaries were not a causative factor. The changes in the other five patients consisted of transient auricular fibrillation and auricular flutter in two, ectopic

beats (nodal, auricular and ventricular) in three and a transient prolonged P-R interval in one patient. Two of these cases also had inverted T waves either in leads I and II or in all three leads. Chest lead studies were normal in the latter case.

Although the blood urea nitrogen was slightly higher in these eight cases than in the uremic patients without pericarditis, in both groups the azotemia was severe, averaging 200 mgm. per cent and 185 mgm. per cent for the two groups. It seems to us probable, therefore, especially in view of the experimental and clinical studies of Schwab and Herrmann[5] that it was the acute fibrinous pericarditis and not the toxic effects of uremia alone which accounted for the alterations in the electrocardiogram mentioned above. However, some unknown factor other than pericarditis must probably account for the occurrence of abnormalities in RS-T sector in but two cases. Schwab and Herrmann believe that these deviations in the RS-T segment in pericarditis are due to ischemia of the heart muscle caused by interference with the coronary blood flow from a rapidly accumulating pericardial effusion. It is, therefore, of more than passing interest that one of our two cases with RS-T deviations had less than 20 cc. of fluid in the pericardium at necropsy twenty hours after the tracing was taken. The other patient had 400 cc. of pericardial fluid. Of the remaining six patients, one showed a normal amount of fluid in the pericardium; three had hydropericardium of moderate amount; two had 700 cc. each. A single tracing in each of the last two cases was normal except for an arrhythmia in one.

PATHOLOGY

As the most important pathologic features of the discussion we have selected the weight of the heart, sclerosis of the coronary arteries, pericarditis and the histopathology of the myocardium. Complete data regarding the heart were available for study in fifty-nine of the sixty-six cases. Fifty-three of these died of uremia and death in the remaining six was due to other causes before renal failure became complete. All of the fifty-nine cases had hypertension. The duration was known to be more than a year in most of the cases and at least five years in a fifth of those dying of uremia.

The cardiac weights of the fifty-three patients who died of uremia ranged from 230 to 650 Gm., averaging 447 Gm., while the average weight of the heart in the six patients who died earlier before renal insufficiency became complete was only 375 Gm. We have arbitrarily used 400 Gm. in the female and 450 in the male as the upper limits of normal weight of the heart. On this basis twenty-seven (51 per cent) of the fifty-three cases of glomerular nephritis with complete renal insufficiency had hearts above normal weight, while only one patient in the nonuremic group was found to have a heart above normal weight. The increase in heart weight was mainly due to left ventricular hypertrophy. Even in those cases whose hearts were within normal limits of weight, 50 per cent showed definite hypertrophy of the left ventricle.

The walls of one or both coronary arteries were definitely thickened in fourteen of our fifty-nine autopsies. Seven of these fourteen died in the fifth decade, two in the fourth, three in the third and one each in the second and seventh. The average weight of these fourteen hearts was slightly less than that given for the entire series. Narrowing of the lumen was present in five cases with an old thrombosis and myocardial infarction in one. The incidence of definite coronary artery sclerosis in this group of glomerular nephritis is practically the same as that given in nonhypertensive heart disease for all ages by Bell and Clawson.[11]

Definite acute fibrinous or serofibrinous pericarditis was present in 48 per cent of the cases dying of uremia. Two cases showed advancing organization of the exudate and almost complete obliteration of the pericardial sac. In the others there were fresh adhesions with varying amounts of pericardial fluid. Hydropericardium of more than 75 cc. was present in twelve patients the largest amount being 700 cc. and the average 300 cc. All had ascites or hydrothorax or both. In seven of these, congestive heart failure played a part. It is significant, however, that in only one patient was hydropericardium present without pericarditis. It is also of interest to note that in these patients with pericarditis there was no inflammation of the other serous membranes except an acute pleuritis accounted for by pneumonia.

The pericardial exudate of uremic pericarditis is almost invariably sterile, and when microörganisms are found they are usually considered to be secondary invaders. Cultures were sterile in all of our patients but one. In this case two types of bacteria (B. coli and a streptococcus) were isolated. These were definitely associated with a superimposed terminal infection of the pericardium. Histologic examination revealed in addition to the mononuclear infiltration of the pericardium commonly present in sterile uremic pericarditis a moderate polymorphonuclear cell infiltration.

The routine histologic study of the myocardium was usually limited to a single block of tissue, unless additional sections were made through areas found abnormal on gross examination. One third of the fifty-nine cases showed slight diffuse interstitial fibrosis, usually unrelated to significant sclerosis of the coronary arteries. Moderate patchy myocardial fibrosis was present in three cases of severe coronary sclerosis. When acute fibrinous pericarditis was

present an occasional case showed slight degeneration and mononuclear infiltration of the myocardial tissue beneath the layer of pericardial exudate. The histologic changes found in the myocardium were, therefore, comparatively unimportant.

SUMMARY AND CONCLUSIONS

1. Clinical and pathological observations have been made on the heart in sixty-six patients who came to autopsy with chronic glomerular nephritis.

2. The heart in this disease is essentially the same as the heart in primary vascular hypertension except for the modifications brought about by the lower average age, the duration of the hypertensive process, the degree of hypertension, and by the terminal uremic pericarditis.

3. The incidence of acute fibrinous pericarditis in fifty-nine patients who died in uremia was 48 per cent, a much higher figure than previously reported in the literature.

4. Our observations on the electrocardiographic changes occurring in uremia suggest that pericarditis is a more responsible factor than the uremia.

5. The frequency of cardiac disorders in the last year of life and the significant rôles they at times play, emphasize again the importance of regarding glomerular nephritis, not as a specific disease of the kidneys but as a generalized vascular and metabolic disturbance in which the extrarenal factors can never be ignored.

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ENORMOUS BENIGN GASTRIC ULCERATION CAUSED BY MULTIPLE FOREIGN BODIES

BY PHILIP H. WHEELER, M.D.*

THE presence of foreign bodies in the stomach has been reported often, but ulceration of the stomach from such foreign bodies seems to be much more unusual. Such reports in the literature are few. Judd and Phillips[1], however, state that they consider ulceration caused by foreign bodies to be fairly common. Among these are two cases of gastric ulceration reported by E. A. Hallas[2], of Copenhagen. One of the cases showed the scar of healed ulcer. He also mentioned two cases, one reported by Tidemand, another by Grauer. The foreign bodies in all cases were cement casts of the stomach. In none of them was the ulceration larger than the size of a two mark piece or pfennig (18 mm.).

A case of an unusually large benign ulceration caused by multiple sharp foreign bodies in the stomach, which entirely perforated the gastric wall without any resulting peritoneal contamination, is reported in this paper. We have been unable to find any such similar case reported in the literature. It is likely that the extreme sharpness of the foreign bodies was a very active factor in formation of the ulcer and it is remarkable that none of these found a way into the abdominal cavity.

The patient, B. T., a former railroad telegrapher, forty-three years of age, white, male, was a mental patient at the Brattleboro Retreat where he had been committed for the second time with a diagnosis of manic depressive psychosis, depressive phase. For a period of several months he had been vomiting occasionally, with increasing frequency. There was coffee ground vomitus with the occasional appearance of bright red blood. The stools were tarry. Careful observation of the patient by attendants led them to believe he was taking foreign bodies into his stomach. X-ray examination, October 10, 1933, by Dr. C. S. Leach at the Brattleboro Memorial Hospital revealed a large irregular opaque mass in the cardiac region of the stomach about three inches by four inches in size which appeared to be made up of metal including staples, tacks, and wire. Staples and tacks were visualized in the right abdominal area in the colon. (Plate 1.) Catharsis delivered staples and tacks. On October 19, 1933, x-ray again showed the gastric mass still present. Two small pieces of metal and a short piece of wire apparently in the ileum and rectum, respectively, were present in addition. Dr. Horace G. Ripley, Superintendent of the Brattleboro Retreat, referred the patient for immediate operative treatment as his vomiting and gastric pain were persisting and his general physical condition was becoming more and more poor.

When examined, the patient was very anxious for relief and freely told of the foreign bodies he had swallowed during a period of three to four months.

*Wheeler, Philip H.—Assistant Surgeon, Brattleboro Memorial Hospital. For record and address of author see "This Week's Issue," page 849.

Before operation he prepared a partial list of objects he had swallowed in order that they "might not be overlooked". Many of the objects such as razor blades, needles, pins, and glass he had covered with gum, food, et cetera. Yet in spite of these precautions he said that his throat had become very sore at times. His admitted intent was suicide, but death in this manner became so hard that he changed to become a very coöperative patient.

Physical examination revealed a moderately emaciated and pale, neurotic patient in some distress from epigastric pain. The teeth, ears, eyes, nose

PLATE 1.

and throat were negative. The lungs and heart were negative to examination. The abdomen showed tenderness to pressure in the epigastrium. No organs or masses were palpable. There was no costovertebral tenderness. Genitalia were normal. Rectal examination was negative. Reflexes were physiological. B. P. 120/80; weight 120 pounds; pulse 80; temperature 98°; respiration 20. The urine showed albumin, slightest possible trace; rbc, rare; wbc, frequent. Blood count showed rbc 4,156,000, hemoglobin 65 per cent, wbc, 21,700. Polymorphonuclears 78, small lymphocytes 19, large lymphocytes 2, eosinophils 1.

Gastrotomy was performed October 20, 1933, and revealed a large stomach with the omentum adherent to the lower third of the lesser curvature. The stomach was opened through a transverse incision opposite this point. Numerous intermingled foreign bodies were removed with forceps and fingers. The entire gastric mucosa, especially along the lesser curvature, was badly lacerated, and friable. The omentum, adherent to the lesser curvature, covered the bed of an ulcer 45 mm. by 32 mm., eroded entirely through the gastric wall. (Plate 2.)

PLATE 2.

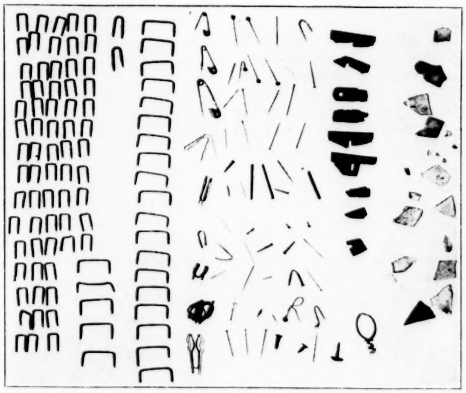

PLATE 3.

Tissue immediately surrounding this was extremely friable. The original gastric incision was extended to permit resection of the ulcer. Closure was made, leaving a drain to the lesser omental cavity.

The pathological examination was made by Dr. Theodore P. Eberhard:

"Sections from four places around the edge show the following: Sharp end of mucosa followed by a thin layer of fibrin and necrotic tissue. The base of the ulcer was composed of granulation tissue and chronic inflammatory cells with complete destruction of the muscularis at some places, and partial replacement at others. The process had spread beneath the muscularis mucosae to the limits of the specimen in some places, and was heavily infiltrated with eosinophils. There is marked activity of the endothelium of the granulation tissue and occasional mitotic figures were seen. No evidence, however, of epithelial malignancy. Gastric ulcer."

Classification of the foreign bodies removed showed sixty-nine narrow staples, twenty-four wide staples, twenty-one pieces of glass, one pencil lead, seventy-two miscellaneous metallic foreign bodies including open safety pins, hairpins, corroded needles, pins, tacks, pieces of safety razor blades, a piece of hacksaw blade, and a screw. (Plate 3.) In addition to these foreign bodies two small nails, two staples, and two pieces of glass in the intestines were later delivered by enemata.

A gastric fistula opened through the drain the seventh postoperative day and closed spontaneously on the twentieth postoperative day. It never gave further trouble. The remainder of the wound healed per primam.

The patient continued to gain after discharge to the Brattleboro Retreat on his twenty-fifth postoperative day until November 13, when he suffered recurrence of pain typical of gastric ulceration. No knowledge was ever obtained of his taking more foreign bodies. A modified Sippy régime was ineffective. The patient was suffering severely, losing weight and strength. At this time he was despondent and attempted suicide. As a last resort he was placed on a course of Synodal. Coincident with the last treatment he became free of pain and commenced to gain. To the present he has remained in good health and at his normal weight. He occasionally has complained of minor gastric discomfort but there has been no vomiting, definite pain, or tarry stools. His mental recovery kept pace with his physical improvement so that he has been allowed vacations and discharges, from which he has voluntarily returned when the stress of society has become too great. He has shown no inclination to resume his suicidal tendencies and has good mental insight.

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ORTHOSTATIC ALBUMINURIA IN HOMOLOGOUS TWINS*

BY HENRY J. BAKST, M.D.,† WINTHROP WETHERBEE, JR., M.D.† AND JOHN A. FOLEY, M.D.†

THAT albuminuria may exist in the absence of any demonstrable pathology of the genitourinary tract is a fact which has been generally recognized since the appearance of Moxon's paper in 1878.[1] Although attention had been called to this matter previously, general acceptance was wanting until that time. Confirmatory reports then followed in rapid succession, with at least three in the same year[2, 3, 4] and innumerable others since that time.

The term "orthostatic albuminuria" was first used by Heubner in 1911. Of the many theories available in regard to its mechanism of production, those of Jehle, Erlanger and Hooker, and Senator have received the widest attention. The first named holds that lordosis of a particular type involving the upper spine (12th dorsal and 1st and 2nd lumbar vertebrae) is responsible for the production of albuminuria because of stasis resulting from interference with renal circulation.[6] Erlanger and Hooker believe that the chief factor in the production of orthostatic albuminuria is a diminished pulse pressure which occurs with the change from the reclining to the erect posture.[7] Senator, however, pointed out that orthostatic albuminuria is indicative of some degree of renal pathology, even though it may not be demonstrable by the methods now at our disposal.[8]

Various modifications of these three main ideas exist. It has been suggested that the condition may be due to a congenital histological defect resulting in increased glomerular permeability[9], or to local acidosis resulting from renal stasis.[10] It also has been proposed that many cases of orthostatic albuminuria have their origin in infection[11], and Janeway pointed out that a postnephritic albuminuria may often be brought out by standing, and that it may be increased by exercise.[12] In addition, it has been noted that orthostatic albuminuria occurs most commonly in a thin individual with poor muscle tone, visceroptosis, vasomotor instability, and low blood pressure. The chief factor here is renal stasis, due to compression of the left renal vein between the aorta and the mesenteric artery. This view is favored by the fact that in lordosis, the aorta is pushed forward, and in visceroptosis, tension is applied upon the mesenteric artery.[13]

In order to establish the diagnosis of orthostatic albuminuria, certain definite criteria must be satisfied. The essential finding is that of albuminuria when the patient is erect, and its absence when the patient is recumbent. In addition, as Thorp and Wakefield have pointed out, the sediment must be free from casts and erythrocytes, there must be no history sugges-

*From the Fifth Medical Service, Boston City Hospital and the Department of Medicine, Boston University.

†Bakst, Henry J.—Junior Visiting Physician, Boston City Hospital. Wetherbee, Winthrop, Jr.—Junior Visiting Physician, Boston City Hospital. Foley, John A.—Visiting Physician, and Physician-in-Chief, Fifth Medical Service, Boston City Hospital. For records and addresses of authors see "This Week's Issue," page 849.

tive of nephritis or nephrosis, and no physical sign commonly associated with nephritis.[14]

There have been many reports of various diseases occurring simultaneously in homologous or identical twins, but a careful review of the literature has failed to disclose any report of the occurrence of orthostatic albuminuria in identical twins.

CASE REPORTS

J. C., aged nineteen, was referred to the Boston City Hospital on May 13, 1935 by his family physician. The patient had applied for training with the Citizens' Military Training Corps, and had been rejected because of the presence of albumin in his urine. He was then referred to the Hospital for an investigation of this finding. The patient had no complaint of any description, and felt well in every respect.

The past history was not remarkable. There was no history of symptoms referable to kidney disease; no nocturia, frequency, hematuria, edema, visual disturbances, headaches, or similar complaints. He had had a tonsillectomy and mastoidectomy at the age of five. He had also had measles, pertussis, and chicken pox during his childhood. There was no history of scarlet or rheumatic fever, sore throats, pneumonia, pleurisy, or joint pains. He did, however, give a history of "bilious attacks" occurring at rare intervals, consisting of headache, nausea, and urticaria, usually lasting about an hour.

The patient's habits were not remarkable. He usually smoked less than a package of cigarettes, drank one cup of coffee, and three cups of tea daily, and used no alcohol.

On physical examination he was found to be seventy-nine inches in height and 163 lbs. in weight. His blood pressure was 120 mm. of Hg. systolic, and 80 mm. of Hg. diastolic. The examination was entirely negative. The eyegrounds were normal. There was no cardiac enlargement, and no murmurs were made out. There was no scoliosis, lordosis or kyphosis.

Laboratory studies revealed no abnormal data. The hemoglobin and blood morphology were within normal limits except for the finding of 4 per cent eosinophils in the differential smear. The blood serology was negative, and the chemistry, including the nonprotein nitrogen, blood urea nitrogen, blood chlorides, total protein, and albumin-globulin ratio were entirely within normal limits, as were also the urea clearance, phenolsulphonephthalein excretion, and urinary concentration tests. The daily urines showed wide variations in specific gravity with occasional albumin in varying amounts, no sugar, and a negative sediment.

X-rays of the kidneys, ureters, bladder, chest, gastrointestinal tract, anteroposterior and lateral views of the spine, and pyelograms were negative.

V. C., twin brother of the above, entered the hospital on the same date, for the same reason. He likewise felt perfectly well, had no complaints of any kind, and appeared to be in good health.

The past history was essentially not remarkable. He had had a tonsillectomy at the age of five, and also had had measles, chickenpox, and pertussis at the same time that his brother had had these illnesses. In addition he had had a mild attack of diphtheria. There was no history of renal disease, rheumatic fever, tonsillitis, pleurisy, pneumonia, or scarlet fever.

His habits varied slightly from those of his brother. He used a considerable amount of tobacco including cigars, pipe, and cigarettes. He was accustomed to drink tea and coffee moderately, but did not drink alcohol in any form.

Physical examination failed to reveal any abnormality save for a soft, blowing, apical systolic murmur which was heard only by one examiner. The heart was not enlarged, the rhythm was regular, and the pulmonic second sound was not accentuated. The blood pressure was 125 mm. of Hg. systolic, and 80 mm. of Hg. diastolic. He was 70½ inches in height, and weighed 155 lbs.

This patient was put through the same laboratory procedures as his twin brother, and, here again, the results were entirely within normal limits. The same x-ray studies were also negative.

The family history revealed several interesting facts. The mother was supposed to have had "kidney trouble" during her second pregnancy (her first pregnancy had resulted in a stillbirth). The pregnancy in question, however, proved to be uneventful, and she went through several subsequent pregnancies which were normal in every respect. Besides the twins, aged nineteen, there are four healthy children, three boys aged eighteen, seventeen, and sixteen, and one girl aged thirteen. The father is well except for the fact that he suffers from hay fever. This was of interest in that one of the twins showed probable allergic manifestations.

The maternal grandfather died of tuberculosis, and the grandmother of childbirth.

The paternal grandfather is living and well, and the grandmother died of cardiovascular renal disease.

One of the boys' aunts had had twins, and a first cousin also had had twins, each on one occasion.

The twins were delivered at the Homeopathic Hospital in Boston on August 22, 1915. One weighed 5½ pounds at birth, and the other weighed 6 pounds. This difference in weight persisted through childhood. They were always very similar in appearance, in fact, they were indistinguishable to people outside the family circle. Their features and bodily characteristics showed a most striking similarity, and while they were on the ward, the nurses and doctors had difficulty in distinguishing them. Their mental reactions were likewise very similar, and their school work had always been of the same quality. Both were right-handed, and were moderately athletic. One (V.) wore glasses for reading, the other did not.

The criteria for the recognition of identical twins mentioned by Leavitt[15] were fulfilled, in that the boys showed a striking similarity in appearance, in mental, physical, and emotional characteristics, had similar birth weights, and birth and life histories. It was not possible to determine whether there had been a single placenta.[16] The general summation of resemblances[17] was so striking as to leave no doubt in our minds as to their probable enzygotic origin.

In attempting to form an evaluation of the renal function in each case, it was felt that in view of a negative history and physical examination, normal blood chemistry and renal function tests, and negative urines save for the presence of transient albuminuria, we were entirely justified in assuming that demonstrable renal pathology did not exist. This point of view was further supported by negative x-rays of

the kidneys, ureters, and bladder, gastrointestinal tract, and dorsolumbar spine, and negative pyelograms. It was further felt that we had not only excluded the presence of organic renal disease, in so far as was possible, but had also ruled out such factors as abnormal kidney position, aberrant ureteral course, and postural scoliosis.

In addition, the diagnostic test advised by Janeway was complied with in each instance.[12] The routine morning specimens from each patient showed the presence of albumin (they had been up and about the ward before passing the specimens). They were then put to bed and specimens were collected at the end of the first and second hours. These proved to be free of albumin. After standing for an hour, in a lordosed position, the patients again voided. At this time J.'s specimen showed a slight trace of albumin, and V.'s a trace. After an hour in bed J. voided a specimen which proved to be free of albumin, while V.'s showed the presence of the slightest possible trace of albumin. An hour later both specimens were albumin free.

SUMMARY

We have attempted to present briefly an instance of the occurrence of orthostatic albuminuria in identical twins, together with some of the views in regard to its etiology, and the accepted criteria for its diagnosis.

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ANOREXIA IN CHILDREN

BY MERRITT B. LOW, M.D.*

THE incidence of anorexia in preschool and school children has been variously estimated to comprise twenty to eighty-five per cent of the pediatrician's practice. Brennemann states that, "It is a matter of common knowledge among pediatricians that somewhere around fifty per cent of children seen in private practice do not eat well." In a scientific investigation made among educated people in a university neighborhood the incidence of anorexia was stated to be eighty-seven per cent. A pediatric colleague with a large office practice in a prosperous suburb estimated it at eighty-five per cent in his practice. A very prominent pediatrician has said that "he paid for his house with anorexia". Capper says, "The complaint of loss of appetite, or unwillingness to eat, not only constitutes the basis of the chief complaint in about thirty per cent of a pediatrician's office practice, but it also forms a chief secondary complaint in other so-called chronic diseases."

A practical working knowledge of what constitutes a suitable diet for children is the common property of all those scientifically or professionally interested in the subject. The average mother of today knows more about the essentials of an adequate diet as expressed in terms of food elements, foodstuffs, vitamins, iodine content, calories, etc., than did the best informed physician of twenty-five years ago. Yet the nutritional results are far from satisfactory. So stereotyped is the monotonous similarity of feeding histories in children that just won't eat that the pediatrician can reconstruct each one ahead of time. The children are not "sick", they have no demonstrable organic illness, and the problem is not merely one of nutrition.

The chief cause of anorexia in children is well known by physicians to be psychic. It undoubtedly comes from the struggle of trying to standardize a product which is fundamentally individual. The evolution of so-called scientific artificial feeding has been a long, devious, and complicated process. During the past fifteen or twenty years, "standard" charts have crept in to our appraisement of the individual. A normal gain in weight is certainly one of the essential attributes of growth in childhood, and is the best single, comparative, objective measure of normal physical development that can be expressed in figures. As a natural corollary have come height and weight charts, and the scale and measuring rod have become ar-

*Low, Merritt B.—Surgical Resident, Children's Hospital, Boston. For record and address of author see "This Week's Issue," page 849.

biters of nutrition. The accumulation of a vast amount of new knowledge concerning an adequate diet has placed the emphasis on the physical to the exclusion of the psychological aspects of nutrition. Stature, weight, and size have come to be intimately connected with, yes, sometimes synonymous with, health, resistance to disease, and many other less tangible but none the less valuable characteristics. The natural effect on modern mothers when confronted by standards has been to proceed to standardize their children, to make them weigh what they should according to height-weight tables. That the etiology of anorexia is often purely psychologic becomes evident when it is found that it is cured by hospitalization, summer camps, boarding school, etc.

Attempting to make children's weight conform to the standards of charts is the principal cause of anorexia. Schultz says, "The environment of the child, particularly its relationship to the mother or the immediate attendant, is probably the largest contributing factor or additive cause in the development of anorexia, and creates often the most difficult problem in the successful and permanent relief of the condition." To the harassed mother of a "problem" child, with its somewhat intangible, difficultly treated "bad habits", we have in the eating of food a most tangible battlefield for the attempt to "reform", and a place to focus attention with an easily evaluated result. Furthermore, the mother easily rationalizes herself into thinking that here is no mere bad habit, but a matter of life and death. Probably the most prominent cause of loss of appetite in an otherwise well child is the attempt to make the child eat a definite amount of food at each meal, day in and day out. As Holt has said, however, "Not what one eats but what one digests, matters."

Summing up then, there are at least three factors which enter into the explanation of this biological phenomenon, the young refusing to eat. First, it is a common trait of human nature to rebel against arbitrary authority. Cameron and others call this "negativism". When a child finds himself confronted by an arbitrarily imposed inflexible system of foods and feeding, he exercises a normal rebellion against a system which allows no choice whatever on his part as to time, place, kind, amount or manner of taking food, and which does not consider whether he is hungry or indisposed at the time. Secondly, it is again a part of human nature to glory in attention. A complacent triumph often characterizes the faces of children as their mothers recite to the doctor the long woeful tale of their refusal to eat. Having been taught by circumstances how to acquire the spotlight, the child uses the tools at his command. Thirdly, it is well known and an established fact that physical discomfort (hunger contractions) is abolished in the presence of emotions. Pavlov, Cannon and others have conclusively demonstrated and explained the dependence of digestion on the mental state. Thus, forced feeding may have a direct harmful effect on the stomach that is not ready to function, and varying degrees of gastric atonia may ensue. Final support for the tenability of the psychic hypothesis of anorexia is furnished by the interesting experiments of Davis, who has shown that infants, six to eight months of age and up, when placed before a varied assortment of simple foods, will invariably select a well-balanced diet for themselves, adequate in every respect; and such babies never become anorexic unless sick. Of course there are other factors in anorexia than the psychic ones mentioned above: fatigue, excessive quantities of a particular food (e.g., milk, the baby sucking on the bottle simply because it is pleasurable), the stresses and strains of modern "in-door" life. In the main, however, the above mentioned are the factors involved in the production of what Brennemann has called an "active immunity" against eating, and, in fact, all discipline.

How, then, are we to cope with the situation? The theory is obvious. First, we must make sure that an adequate diet (as regards quantity and quality) is provided, and, secondly, no child must ever be forced to eat (with rare exceptions, as in typhoid fever, etc.). The elimination of organic disease (focal infection, anemia, chronic pyelitis, tuberculosis, syphilis, nasal obstruction, carious teeth, etc.) must be insured. It is probably wise to take the history in the absence of the child. "Tonics" of various types undoubtedly have their place, if only for the psychological effect. Ofttimes, when there is a "medicine", the mother will pay less attention to the eating, at least from the active standpoint. The intimate relation between mother and child from the start as regards nutrition is a fertile breeding-place for all the tribulations that arise from anxiety, and any relief from anxiety and focussing attention often works wonders. The importance of the anxiety-factor in the parents cannot be overstressed as regards all child-behavior problems, feeding, enuresis, and many others. A preliminary period of starvation is recommended by some. Small doses of insulin have been tried, but this is not often a happy choice. The "feeding habit", the well-defined periodic desire or need for food occurring several times a day which is found in all healthy animals, must be cultivated. Attention must be given to taste and appearance of food, and eating between meals must be forbidden. The regulation of all bodily habits must be attained. A just system of reward that bears no connection with bribery seems warranted, at least in many instances. It is im-

portant to remember that caloric requirements (i.e., calories per pound of body weight) decrease with the age of the child. A word of caution must be inserted here in regard to the so-called "bitters". Cushing and Carlson say, "We conclude that in therapeutic quantities the bitters, acting in the stomach alone, have no effect on gastric tonicity, or the gastric hunger contractions, or on the parallel sensation of hunger. In greater than therapeutic doses the bitters inhibit the hunger contractions and abolish the hunger sensations in direct proportion to the intensity and duration of the stimulation of hunger contractions and hunger sensations." The conclusion is thus that the beneficial effects from the use of bitters are largely or entirely psychic (as indeed, is much medicine), and the prescriber is much more important than the prescription.

One cannot fruitlessly nag or force a child to eat without losing that wholesome spontaneous discipline that alone leads to a normal behavior reaction. It is important to make few requests and have them obeyed, rather than many which are not "followed through". Cameron suggests that the best way to combat the child's "negativism" is often to appear to the child as determined to prevent him from eating too much, and not as eager to see him eat well. The mother's duties in regard to her child are therefore (as summed up by Sweet): (1) that she provide the child with regular meals; (2) that the meals be of good quality; (3) that the child shall come to the table with clean hands, so he may handle his own food; table manners should not receive too much attention, because eventually the child will copy the manners he sees, not the ones he hears about; (4) that he shall remain at table a definite length of time, for example, thirty minutes; (5) food must be served without one word being said about food. Food is of and from the parent; feeding is solely within the province of the child. We must let hunger lead to appetite and appetite urge on to the acquisition of food. There is no greater joy in life than to have urgent, earnest desire satisfied as the results of one's own efforts. Self-satisfaction is one of the true rewards of all endeavor. We must therefore allow our children the privileges of hunger and the joy of appeasing it.

In conclusion, as elsewhere prophylaxis seems to be the most practical key to the problem, as far as future progress is concerned. We are, furthermore, privileged to work in this disease with the brightest type of child, the sensitive, high-strung, slightly neurotic child who is to be the standard bearer and pioneer as an adult. New health standards in addition to height, weight, and gross size must be drawn up and borne in mind. Examples of these might be posture, nervous stability, reactions to environment, resistance to disease, blood picture, intelligence, etc., difficult but very important standards to set. The importance of detail in the upbringing of babies cannot be overly stressed. Six to eighteen months is the crucial age. Feeding from the spoon should probably be started at one or two months. Just because the infant likes to suck, he should not be given excessive amounts of milk from the bottle between six and twelve months of age. No bottle should be indulged in after a year. The child should begin to feed himself at fifteen to eighteen months. Above all, we must bear in mind that the child has an individuality of his own which is entirely different from the individuality of adults. There is something biologically primitive about him that cannot be rapidly transformed into the more stereotyped requirements of adult life, and particularly of a confused "civilization" that is now moving so swiftly that even the adult is often lost trying to keep going. In growth and mental and physical expansion, there is a delicate balance between direction and self-expression, between convention and originality, between regulation and the fostering of initiative, between having one's problems solved and solving them, between prohibition and freedom, between the acceptance of the old and the desire for the new.

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VERMONT STATE MEDICAL SOCIETY

CHEMISTRY IN RELATION TO THE PRACTICE OF MEDICINE*

BY C. F. WHITNEY, M.D.†

A GENERATION ago the necessity of the study of chemistry as a preliminary to the practice of medicine was doubted by many, and even now medical students ask what is the relation of biochemistry to medicine. Prior to the last century, due to the dearth of known facts, chemical thought was largely limited to speculation. Having its origin, like the practice of medicine, in magic, explanations were largely fantastic rather than rational. Chemistry has made great strides during the last fifty years, and one can assert without fear of contradiction that the elimination of superstition, of useless remedies and foolish practices, and the substitution of a rational treatment may be credited largely to the close and detailed study of the exact sciences, such as chemistry and physics.

A recent addition to the chemical armamentarium is the branch known as physical chemistry. Due to the fact that most reactions in the body are those involving surface phenomena and ionic dissociation in an organism made up largely of colloidal particles, one realizes the importance of a knowledge of surface tension, colloidal dispersion, interfaces, salt equilibrium, aggregation and dispersion, diffusion, viscosity, oxidation and reduction, x-radiations, radioactivity, high tension and high frequency phenomena, electrical potential, optics, etc.

Physiology, pathology, and immunology could never have even distantly approached their present value were it not for the stimulus of chemical ideas and facts, nor could the field of nutrition have advanced to its present position without the help of chemistry. Formerly one spoke in vague terms of stimuli passing from nerve to nerve through nerve fibers like the wires of a telegraph system. We now know that this impulse is transmitted from nerve fiber to nerve cell by liberation of a minute charge of acetyl choline.

The weight of this substance used to transmit a single nerve impulse to a single ganglion cell is so minute as to be about equal to 10 to the minus 21st power in grams, or 20 ciphers to the right of the decimal point followed by the figure one. Therefore, in precise chemical terms we seem soon to be able to describe the transmission of effects from nerve fibres to reception cells in the whole peripheral nervous system. The central nervous system has not been explored in the same way and remains a deep complex problem.

In the work on vitamins, sometimes called exogenous hormones, and on hormones, rapid strides are being made. Vitamins C and D have been artificially prepared in the pure state, the chemical nature of A is known and the pure crystaline vitamin B is available commercially.

The hormones are produced in definite parts of the body and carried by the blood and lymph to other parts where changes are effected. The estrus producing hormone which is excreted in the urine in pregnancy can be extracted from the urine in large quantities by benzene and crystallized. This substance is now known to be one of a group of condensed ring compounds consisting of a phenanthrene molecule fused to a 5-membered ring and common to the organic compounds occurring in the body. Methods for separating the different principles of the pituitary lobe, appropriately called the conductor of the endocrine orchestra, which presides over the sex-gland activities, normal growth, and in some ways seems to antagonize the overaction of insulin, are already being used. The chemical structures of epinephrine and thyroxin are now known and they are being prepared by artificial synthesis. Likewise the chemical structure of one of the female sex hormones is known and synthetic production will undoubtedly be made soon. Insulin is also obtained at the present time in crystalline form, though its chemical structure is not known.

The body is essentially organic in nature with 70 to 80 per cent water. It consists of proteins, fats (lipins), carbohydrates and mineral substances. All parts except the bones are of a fluid to plastic solid consistency, each maintaining its physical condition essential for proper functioning by chemical combination of very intricate and as yet little known nature. The mineral substances such as sodium, potassium, calcium, phosphorus, iron, iodine, and sulphur are perhaps the simplest when considered by themselves, but in the body they are in organic combination and each absolutely essential for normal physiological action. Sodium and potassium, though very similar chemically, cannot replace each other, both being essential. Calcium and phosphorus in definite ratio and with the aid of vitamin D are essential for proper bone growth. The former is also necessary for blood clotting and the latter for the metabolism of carbohydrates, fats, mus-

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†Whitney, C. F.—Professor of Physiological Chemistry and Toxicology, University of Vermont. For record and address of author see "This Week's Issue," page 849.

cle contraction, etc. Iron is necessary in hemoglobin and acts as a catalyst for internal oxidations. Iodine is found in the thyroid gland and sulphur is necessary for growth and development. This is a brief review of the value of the most important inorganic constituents. A few others, including copper, are necessary, but less is known of their value.

The carbohydrates used in foods are mostly confined to starches and the common sugars, which after digestion are absorbed into the blood as single sugars and stored as glycogen, circulate in the form of glucose, or are converted into fat. The chief fatty foods are the well-known common fats. These are absorbed and circulate in the blood as simple or compound lipins and later they are used to produce energy. Proteins are much more complex and form huge molecules. The molecular weight of hemoglobin which is one of the smallest is about 70,000 as compared with glucose which is 180. Proteins are made up of amino-acids of which there are some twenty-two or twenty-three. All proteins are broken down into these comparatively simple bodies (the largest having a molecular weight of 190), before absorption into the blood. It is these substances rather than their combination in the form of protein which interest the physiologist and upon which the well-being of the individual depends. Fortunately the body can manufacture most of the amino-acids but about a half dozen cannot be so formed, and for that reason must be taken as such in the protein complex.

The forms in which substances are excreted are well known, but the great and undiscovered chemical changes are those which occur between absorption and elimination. This is a most attractive and fertile field which will gradually unfold chemical changes of inestimable value to the physician. As an illustration of the chemical changes known to take place in the body one might mention those that occur in the red blood cells in the process of internal respiration. When oxygen enters the capillaries of the lungs as the result of increased oxygen pressure, it combines with hemoglobin thereby producing a stronger acid. This robs the carbonate salt of its base converting it into carbonic acid, and allows the passage of carbonic acid gas into the alveoli. As the blood reaches the tissues the pressure of gases is the reverse of that in the lungs. Oxygen leaves the hemoglobin to enter the tissues, and carbonic acid gas enters the blood. Neither oxygen nor carbonic acid gas can be carried in any appreciable amounts in simple solution, but must be combined in the form of salts. The reduced hemoglobin, being a weaker acid than oxyhemoglobin, will give up the base taken over in the lung capillaries by the oxyhemoglobin and this base will be available for union with the carbonic acid gas. This exchange of gases is greatly aided by the passage of chlorine, and to a much less extent the acid radicals of phosphoric and sulphuric compounds, into and out of the red cell. Only negative ions are allowed to pass, positive ones being barred. This knowledge explains one of the chief mechanisms which regulate the acid-base balance of the body, changes which if equal to the difference between the hydrogen ion concentration of tap and distilled water would be incompatible with life.

The arbitrary ways in which physicians in the past have outlined diets for their patients have often been unfortunate; allowing this and excluding that, red meat and white meat, no fruit juices with milk, no protein in nephritis or high blood pressure, etc. Even now there are those who do not realize that most fruit juices owe their acidity to organic acid salts which leave an alkaline residue after metabolism, and so tend to reduce acidity rather than increase it. Many things of surgical and medical value were learned during the World War. Among them was the cause of "war edema" prevalent in prison camps, the underlying cause being protein deficiency. The edema associated with nephrosis is due to a lowering of the osmotic pressure of the plasma. Of the two proteins, albumin and globulin, it is the former that is excreted in greater amounts, has a greater osmotic pressure, and is less easily regenerated. Blood analysis to determine the amount of these proteins in cases of edema may therefore aid in determining the cause and treatment.

Psychochemistry is the name applied to that branch which particularly applies to the central nervous system. In paranoia, dementia praecox, epilepsy and manic-depressive psychoses, chemistry is contending with histology in conveying the more knowledge regarding the fundamental changes. Chemical examination indicates a deficiency in catalytic iron and neutral sulphur in some of the mental disorders. These may be inherent deficiencies which cannot be corrected, but knowledge of these changes may aid in finding the remedy. Dementia praecox may yet be found to be a deficiency disease as much as rickets or scurvy, and as amenable to treatment, the required adjuvant being in this case oxygen. This deficiency may be due to dietary deficiency, production of toxins or some abnormal nervous interference with proper oxidation. In this last connection one is led to believe that the beneficial effects of amytal may be the removal of some inhibitory action upon normal oxidation processes.

We hear lately of the rôle of the water balance in epilepsy and of alterations in electrical potential and colloidal dispersion in the manic-depressive psychoses. So long as these different physicochemical phenomena function nor-

mally between narrow limits there is no disturbance, but let an unbalance occur sufficient to disturb the equilibrium in oxidation-reduction, aggregation and dispersion, hydration and dehydration, electrical potential or sodium-potassium and calcium-magnesium ratios and a disordered functioning of the brain will result. It has long been known that the metabolism of the brain is very active, no tissue needs more oxygen and no cells will be so quickly destroyed by lack of oxygen. The psychic phenomena shown in partial deprivation of oxygen as in carbon-monoxide poisoning and partial suffocation or to increase atmospheric pressure as in caissons, are well known. Cytochrome and glutathione, besides being essential catalysts for oxidation with iron and other metallic substances, are comparatively abundant in the brain.

Many mental conditions seem to be affected by water balance. Particularly is this true in epilepsy. Free water in tissue spaces holds sodium, and in the cells potassium. Water is also bound in the cytoplasm and nucleus. Fevers can be produced by dehydration, and the delirium of fevers is attributed to increase in bound and decrease in free water. The convulsions and coma of water intoxications are due to increase of interstitial water, and through osmosis can be relieved at once by intravenous administration of concentrated saline. It is well known that the size of colloids changes with absorption of water which latter is influenced by contact with acid. It is thought that the explanation of epilepsy rests in the hypothalamus which somehow has some control over water metabolism and the tone of blood vessels.

Much research work has been done on the biochemistry of cancer. Studies have been made of the chemical constitution of tumor tissues, blood analysis in tumor cases, and the metabolism of these growths. Of these three, the most hopeful results come from the metabolism studies. Cancer tissues seem to have a high sodium and potassium and a low calcium content. It is more hydropic than normal tissue and the nitrogenous constituents differ from that of normal. Much speculation has resulted from the chemical findings, such as causation based on salt unbalance with its effect on cell permeability, and treatment by administration of calcium salts. On the whole, however, little positive help has been gained from chemical analysis of cancer tissue. Blood analysis indicates that the alkalinity of cancer patients is higher than normal but this also is true of a number of maladies; so that little of practical importance has been found in such analyses. In metabolism experiments, it has been found that tumor tissue utilizes an excessive amount of carbohydrate and produces an excessive amount of lactic acid. Tumor tissue is able to utilize very little oxygen in comparison with normal tissue. For this reason it has to obtain sufficient energy for growth by breaking down an excessive amount of glycogen to lactic acid. This is very different from muscle, for example, which is able to form lactic acid from the hexosephosphates. These differences between the metabolism of cancer and normal cells seem to be fundamental and of great importance in the study of the cause of cancer.

In this brief discussion certain phases of the subject familiar to all physicians, particularly those having intimate access to clinical hospital laboratories, have been omitted. These would include such subjects as gastric, urine and blood chemistry, liver and kidney function and metabolism tests. Nor has any special mention been made of the discovery of insulin, and only a brief mention of the vitamins and hormones. It might be mentioned that the determination of blood cholesterol is of assistance in confirming a diagnosis of nephrosis or myxedema or to determine the severity of diabetes; the icterus index test in cases of anemia or jaundice; the blood uric acid estimation in cases of suspected gout; the blood calcium in spontaneous fractures, bone softening, generalized osteitis due to hyperparathyroidism and in tetany of unknown origin; the inorganic phosphorus of the blood in cases of rickets or infantile tetany, and blood creatinine when the blood urea is over 30.

DISCUSSION

PRESIDENT MARSHALL: In regard to this excellent paper a good deal of thought was given as to whom we should have discuss it, and after considerable deliberation it was decided to ask Dr. Paul K. French, of Burlington.

DR. FRENCH: Dr. Whitney's paper has opened up for me an unthought of field of speculation. He has stated that:

> (1) "So long as the different physico-chemical phenomena function normally between narrow limits there is no disturbance of the brain, but let an unbalance occur sufficient to disturb the equilibrium in oxidation-reduction, aggregation and dispersion, hydration and dehydration, electrical potential or sodium-potassium and calcium-magnesium ratios, and a disordered function of the brain will result."
>
> (2) "That no tissue needs more oxygen and no cells will be so quickly destroyed by lack of oxygen.
>
> (3) "It is thought that the explanation of epilepsy rests in the hypothalamus which somehow has some control over water metabolism and the tone of the blood vessels."

He has spoken of the fever produced by dehydration and suggested that delirium convulsions and coma may be due to an increase in bound and a decrease in free water, mentioning the immediate relief of the convulsions of water intoxications by the intravenous administration of concentrated saline.

As we think over these statements and their application to the mental and physical brain disturbances,

which we as physicians are continually seeing, may not an understanding of the chemical relationship and balances present in the brain point the way to relieving symptoms by seeking a means of restoring the circulation and metabolism of the tissue to normal? A better understanding of the cause should afford a more successful approach toward relief.

I feel that this paper is going to help me to study, from a chemical viewpoint, the symptoms of my patients, and I believe it can be of real help.

All I could do in the length of time afforded was merely to touch on these things, and I hope this paper will make us think of their relationship so that we will study it out for ourselves.

PRESIDENT MARSHALL: Dr. Dalton.

DR. DALTON: When you ask me about this subject you have something I don't know a thing about. I studied my chemistry a long time ago, and there was not anything applicable to what Dr. Whitney has said, and I am entirely unable to discuss this paper.

PRESIDENT MARSHALL: This paper is quite technical for most of us. Dr. Whitney, have you something to say in closing?

DR. WHITNEY*: When I studied medicine I had chemistry, and as I recall I think the only technical procedures that we did were gastro-analyses and urinalyses. At the present time they pay no attention, practically, to either of these, but to the fundamental chemistry so that the student will learn to interpret phenomena and, through chemical means, realize the great importance of chemistry. This physical chemistry is very important, and the knowledge is much more important than it was thirty years ago when I studied medicine. We are often criticized because we do not teach students, in the first year, how to analyze urine, but we do metabolism tests and try to get at the basis and fundamental rather than simple tests.

*Dr. Whitney's closing discussion has not been edited by him.

MISCELLANY

POSTGRADUATE FELLOWSHIPS AVAILABLE TO MEMBERS OF THE VERMONT MEDICAL SOCIETY

Under the Division of Public Health of the Commonwealth Fund, the office of which is at 41 East 57th Street, New York City, opportunities are offered to the members of the Vermont State Medical Society to receive postgraduate instruction at the Harvard Medical School, available to at least eight members.

The following statement is from the Commonwealth Fund indicating the nature of the course and the necessary qualifications.

WM. G. RICKER, M.D., *Secretary.*

Commonwealth Fund fellowships available, in the subjects indicated below, to members of the Vermont State Medical Society given at the Harvard Medical School, Courses for Graduates, 25 Shattuck Street, Boston, Mass.:

Medicine, given at the Massachusetts General Hospital, Peter Bent Brigham Hospital or Boston City Hospital. A group of at least six must take the course at one time. Such a group may be made up from any one of the four states in which the fellowships are offered, namely, Vermont, New Hampshire, Maine or Massachusetts.

Pediatrics, course is given at the Children's Hospital. Not more than two may take the course at one time. Fellows live at the hospital.

Obstetrics, courses given at the Boston Lying-in Hospital. Not more than two may take the course at one time. Fellows live at the hospital.

Office Surgery, course is given at the Boston City Hospital; designed for physicians engaged in general practice; subjects studied are surgical problems met in the office; instruction in the out-patient department. A group of at least six must take the course at one time.

Fellowships are for one month. Preference will be given those who take the course in medicine, for a second month in medicine or in obstetrics, pediatrics or office surgery, when fellowships are available during succeeding years. The stipend is $250.00 plus tuition and traveling expenses from place of residence to Boston and return.

Qualifications: Applicant must be a graduate of a grade "A" medical school; a member of the Vermont State Medical Society in good standing; must have been in practice at least five years and should preferably be under forty-five years of age; and must be a resident of a community of less than 10,000 population. Application blanks may be obtained from the Commonwealth Fund, 41 East 57th Street, New York City, or from the Secretary of the State Medical Society.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22171

PRESENTATION OF CASE

A sixty-eight year old white Russian watchmaker was admitted complaining of constipation and bleeding from the rectum.

Six months before coming to the hospital the patient first noted that his bowel movements, which had previously occurred regularly once daily, became lessened in amount and frequency. The stools gradually became narrow and finger-like in character. For about two months he passed small amounts of dark red blood by rectum, and at the time of admission each bowel movement contained one to two tablespoonfuls of blood. During the three months preceding his entry he developed increased frequency of micturition associated with some burning dysuria. During the past one and a half years he suffered cramp-like pains in his right leg whenever he walked rapidly. For fifteen years he had recurrently a sensation of substernal oppression following exertion or excitement. This had recently become much less frequent.

Physical examination showed a fairly well-developed and nourished sallow man in no discomfort. The lips were somewhat cyanotic. The chest was said to be emphysematous in configuration, and expansion was limited. The breath sounds exhibited a prolonged expiratory phase. Occasional expiratory wheezes and musical râles were audible. The heart was not enlarged to percussion. The sounds were loud, regular, and a prolonged systolic murmur was audible from the apical to the aortic area. This was transmitted into the neck vessels. No thrill was palpated. The pulse was said to exhibit a slow rise, long plateau, and slow fall. The blood pressure was 190/85.

The temperature was 98°, the pulse 75. The respirations were 22.

Examination of the urine was negative. The blood showed a red cell count of 4,890,000, with a hemoglobin of 80 per cent. The white cell count was 9,700, 68 per cent polymorphonuclears. A stool examination showed a two plus reaction to the guaiac test. The nonprotein nitrogen of the blood was 38 milligrams per cent.

On the fourth day a first stage combined abdominoperineal operation for carcinoma of the rectum was done. The patient reacted well postoperatively but on the second day he began to complain of a sensation of pressure over the entire anterior chest. At this time the nurse noted that his pulse was irregular and had increased to a rate of 100 to 140. On the morning of the following day there was sudden increase in pain over the sternum and the anterior right chest, and the patient appeared cold and clammy. The lips and hands were markedly cyanotic, and the respirations were labored. The pulse was 140 and grossly irregular at the apex. The blood pressure was 110 to 130/75. The pain became more intense and radiated down the left arm. Examination showed that the right chest was full of coarse râles. An electrocardiogram exhibited auricular fibrillation with a rate of 170. There was inversion of T waves which was said not to be diagnostic of recent coronary occlusion. He had received about six grains of digitalis during the thirty-six hours preceding his initial discomfort. On the following day the lungs were clear and there was a squeaky murmur at the apex of the heart. No details were noted. The temperature, which had risen to 101.6° on the day succeeding his operation, fluctuated thereafter between 98° and 102° for about a week. Two days after the acute attack the pulse was recorded as being regular, slow, and of good volume.

Two weeks after the initial operation a posterior excision of the rectum was done. On the following day his temperature, which had been moderate for the preceding week, rose to 101°. Thereafter for the remainder of his hospital stay it fluctuated between 98° and 100°. Two days postoperatively, having received 300 cubic centimeters of an intravenous infusion, the patient suffered a sudden attack of substernal pain. The quality of the pulse remained good and its rate was unchanged. Thereafter there were several minor attacks of precordial distress, but on the twenty-sixth day after the second operation he again suddenly developed severe substernal pain. Morphin was administered with some relief. He was found at this time to have a rather marked degree of pyuria and constant vesical drainage was instituted. He became progressively weaker, however, and died nine days after his last acute attack, on the fifty-third hospital day.

DIFFERENTIAL DIAGNOSIS

DR. PAUL D. WHITE: I think it is of some added interest to know whether this patient was a Hebrew. We do not know the incidence of disease in the various groups that make up Russia, but we do know that cardiovascular disease is very common in Hebrews who have come to this country from Russia.

Apparently his chief complaint was this difficulty with the bowels, giving evidence of a spastic state, bleeding, and local bladder irritation, which, as it turns out later, were the result of a rectal carcinoma.

"During the past one and a half years he suffered cramp-like pains in his right leg whenever he walked rapidly." This of course suggests intermittent claudication, which commonly occurs in both legs but may be limited to one leg.

"For fifteen years he had recurrently a sensation of substernal oppression following exertion or excitement." This sounds like angina pectoris of long duration, fifteen years. Its decrease may have been the result of the inability to walk because of the intermittent claudication. That is all there is in the past history. I would like to know many other things, particularly the physical examination in the past in view of what is to follow. It is quite likely that he had consulted a physician for the intermittent cramp-like pains in the leg and chest and it would be helpful to know what had been found on cardiac examination. We are prone too often to neglect such clues, which may be important and the chief aids to correct diagnoses.

We have then this abnormality of the lungs which may be on the basis either of chronic bronchitis with emphysema and asthma, or of left heart weakness. The cyanosis of the lips may be associated with that condition of the lungs or it may be local. Sometimes lips and fingers are blue due to peripheral or local stasis and not to cardiac or pulmonary disease.

"The heart was not enlarged to percussion." Of course such a statement is entirely unreliable in this case because of the pulmonary condition which prevents one from outlining proper heart borders. If the apex impulse could not be felt we would like to have x-ray evidence, but there is no report of such. There should have been some statement as to whether or not the apex impulse could be felt.

From this description I should judge that this murmur was a single murmur, not composed of two separate murmurs, one at the apex and one at the aortic area. It probably sounded the same all over the precordium. It was loud and transmitted into the neck. One of the most likely causes of such a murmur is aortic stenosis. A murmur that is loud and well heard both at apex and aortic area is much more likely to be aortic than mitral. A mitral-systolic murmur is not well heard in the aortic area but an aortic systolic murmur is always well heard at the apex, especially if due to aortic stenosis. There has not been enough appreciation about these points of location and transmission of systolic murmurs. It is conceivable, however, that there may have been separate aortic systolic and mitral systolic murmurs that have not been differentiated in this record. There is no evidence of a diastolic murmur or of a systolic thrill.

The character of the pulse is certainly important and this description, if not misleading, has probably been put in to give us a further important clue. It closely fits the description of a plateau pulse such as is found with aortic stenosis.

"The blood pressure was 190/85." In other words a definite hypertension which is superimposed on whatever else is present in the heart and makes us wonder if the systolic murmur heard over the aortic area may not be due to aortic dilatation after all, but we should remember that the blood pressure may be high in the presence of aortic stenosis. Evidently the aortic second sound was present. It would probably be mentioned as absent if it had not been heard.

The laboratory examination of the urine reveals no important renal or bladder infection at this time. There is no important anemia. The nonprotein nitrogen of the blood was normal.

"On the fourth day a first stage combined abdominoperineal operation for carcinoma of the rectum was done." Evidently it was hoped that the carcinoma of the rectum might be removed and that the risk which was considerable in this case was worth taking. He was evidently in poor health on the basis of the long history of angina pectoris, of the cardiac findings, and of the pulmonary findings. These are three points which made him an unfavorable risk for operation. There is no note of what anesthesia was used, but that is almost always less important than the skill of the anesthetist.

The sensation of pressure over the entire anterior chest would indicate the probability of auricular fibrillation starting at that time. A pulse is rarely irregular at a fast rate unless there is auricular fibrillation.

"The pulse was 140 and grossly irregular at the apex," more or less as it had been before, but now somewhat faster, sustained at 140 rather than 100 to 140. There was a marked drop in pressure.

"There was inversion of T waves which was said not to be diagnostic of recent coronary occlusion." I expect the inference is that there was no great change in the S-T interval, that is the level where the T comes off from the Q-R-S, the shape of the T wave was not indicative of coronary occlusion. That is of considerable importance.

"Two days after the acute attack the pulse was recorded as being regular, slow, and of good volume." This is confirmatory, then, of the diagnosis of paroxysmal auricular fibrillation. At the beginning we noted the irregular pulse and sensation of pressure over the anterior chest. What may we say about that? I believe it is impossible on this evidence alone to tell whether the auricular fibrillation was secondary to his

coronary condition or vice versa. A number of times coronary thrombosis has been diagnosed in a patient with prolonged anterior chest pain and paroxysmal tachycardia without coronary thrombosis. Paroxysmal tachycardia or paroxysmal auricular fibrillation with a rate as fast as this can in itself produce angina pectoris, a kind of status anginosus. That is probably the situation here in view of the fact that he had had angina pectoris on effort for the previous fifteen years. He had narrow coronary arteries or at least an insufficient coronary blood supply. The narrowing of the coronaries in itself may not have been sufficient. We may have in addition aortic stenosis which would reduce still further the volume of the coronary circulation. The status anginosus due to paroxysmal auricular fibrillation with much narrowing of the coronary arteries and some aortic stenosis might explain the beginning of this postoperative complication, but we must assume that there is some other complication, too, such as congestive failure, if we take into account the increased respiratory difficulty, the cyanosis, the signs in the chest and the râles in the lungs. It is not likely that congestive failure or the status anginosus alone, or combined, can do all this, because we have fever that lasted a week.

Pulmonary embolism is a common postoperative complication, especially in a patient who has had cardiovascular disease. Could this not have occurred here, complicating the paroxysmal auricular fibrillation and angina pectoris? It certainly is very suggestive, especially because of the fact that only one lung, the right, was involved. There is no note of any abnormality of the left chest. The right chest was filled with coarse râles. Did he have pulmonary embolism with infarction in the right chest complicating the paroxysmal tachycardia? It seems to have come on the second postoperative day. On the first postoperative day there was simply the rapid irregular heart action with pain; on the next day there was a sudden increase of pain over the chest combined with a state of shock, cyanosis, drop in blood pressure, and fever. The other two possibilities that could explain such a state of affairs would be complicating infection in the right lung and coronary thrombosis

The electrocardiographic evidence is against acute coronary occlusion. Also, the localization of the râles in the right lung, unless he were lying on his right side all the time, is against coronary thrombosis with secondary congestive failure. We would expect râles at both lung bases with failure sufficient to give rise to cyanosis and dyspnea.

The paroxysmal auricular fibrillation ceased two days after the acute attack began and several days before the temperature dropped back to normal, so that toward the end of that week there was no longer any effect from rapid cardiac action. You will notice that the heart was said to show a squeaky murmur at the apex when the lung cleared. That murmur was undoubtedly the remains of the loud systolic murmur that had been heard when he first came in, in better condition. This state of prostration in which we find these patients is an important reason why aortic stenosis has frequently been missed clinically in a very sick patient.

We may assume that this recurrence of pain came at the time of the intravenous infusion of 300 cubic centimeters. It is possible that angina pectoris was induced by the increased heart work in taking care of that extra amount of fluid, a thing that does happen in patients who are very prone to angina pectoris. There is no note as to what his pulse rate was at that time or of further electrocardiograms.

"He was found at this time to have a rather marked degree of pyuria and constant vesical drainage was instituted." Thus we have a still further complication of pyelitis or cystitis, making it more difficult for him to weather all these storms.

We would like to know whether his death was sudden. Was this terminal illness due to coronary thrombosis or pulmonary embolism or infection in a patient with a weak heart who had been through these two operations? Too many cards were stacked against him. I do not believe it is possible to tell from the few notes at the end of the record what the terminal event was.

I prophesy that we will find in this man a moderately enlarged heart (in spite of the statement at the beginning that the percussion failed to show any enlargement), secondary to two factors, hypertension and, I believe, also some aortic stenosis. Coronary artery narrowing will be present, on the basis of atherosclerosis. Coronary thrombosis is a possibility but there is no proof of it. I expect the aortic valve will be calcified. Paroxysmal auricular fibrillation and angina pectoris were functional events of importance. He also had carcinoma of the rectum, cystitis or pyelitis and probably pulmonary infarction. As for the cause of aortic stenosis if present, it is in just such a patient that aortic stenosis may be discovered, usually without a clear history of previous attack of rheumatic fever. Sometimes examinations ten to twenty-five years before give evidence of a murmur such as is described here. I have recently seen a man whom I examined first nine years ago, at the age of fifty-three, with a very loud murmur heard over the whole heart, maximum at the apex, and slight enlargement of the heart. I made a diagnosis of cardiac enlargement with mitral regurgitation of unknown cause. Last fall he had attacks of pulmonary edema and on examination he then showed an aortic systolic thrill and serious heart failure. It is possible

that our present case did not have aortic stenosis but certainly that diagnosis would explain best the original cardiac findings as noted in the record at the beginning of his stay in the hospital.

DR. TRACY B. MALLORY: This case obviously had two aspects, the medical and surgical. We chose to make the medical side the diagnostic problem, but we would like to hear about the surgical side.

DR. DANIEL F. JONES: I do not know what I can say about this surgically except to tell you that I think that anyone undertaking an operation for carcinoma of the rectum in a man with a heart like this is taking his reputation in his hands and at least trying to throw it away; but I have no such feeling about these cases. If it is possible to get rid of carcinoma of the rectum even at considerable risk—this was a very large lesion and he was very uncomfortable with it—I have no hesitation whatever in trying it. We nearly succeeded in this man. The strange thing is that he stood the abdominal part of the operation very well. He had ether during that time but I agree with Dr. White that it makes a great deal of difference as to whom you have to give the ether. I am quite alone on this question, except for one man, and that is Gabriel in London, he and I approve of ether in these cases. The patient had spinal anesthesia with the second stage because we are able to give a small dose and give it low.

There is nothing in regard to the diagnosis that I can speak of here. There is not enough history. There is one thing that I disapprove of very much, and that is the statement that the stools gradually became narrower and finger-like. I think that is a pretty uncertain thing to go by. The books all have a much more picturesque way of talking about it; that is, the "ribbon stool" but I think it is about as poor a symptom to go by as anything can be, because if you know the structure of the sphincter, you know it is oval, and if the bowel movements are soft you will get a ribbon stool or with any soft movement you may get a narrow finger-like stool. Consequently I do not think that of much value.

As to diagnosis, there is only one thing I can say and that is he should have been examined six months before he came to this hospital. If you want to know why, there is a very simple reason. He should have been examined because he has had a change in bowel habit and sensation, and that is all that any general practitioner or medical man needs to know. I do not expect a general practitioner or medical man to make a diagnosis of carcinoma of the rectum every time, but if there is a change in bowel habit or sensation then somebody who can make a diagnosis should be consulted.

This case shows very well that you have to be careful, too, in these heart cases. He stood the first operation very well and we let him go for two weeks in fairly good condition. It was getting along and we had to operate. After the second operation he did not do so well because he had a load put on him which he could not get rid of and consequently went downhill. He had what you would expect, some infection. It was a very large wound, and many of these cases have temporary paralysis of the bladder as they must have drainage of the bladder; therefore, you must expect some infection of the bladder. He had nothing to indicate any pyelitis. As to lung emboli, an interesting fact is that in spite of the great extent of this operation and the fact that many pelvic operations are likely to cause lung emboli these rectal cases very rarely for some reason or other have lung emboli. I do not know why it is.

DR. DONALD S. KING: The day after operation this patient had an elevation of temperature, pulse and respirations. I was asked to see him with the question of a postoperative pulmonary complication. There were râles throughout the right side of the chest but no definite evidence of pneumonic consolidation or collapse. In view of the story, however, I felt that there was probably beginning postoperative atelectasis. I saw him again the following day, and in the meantime the patient had had the acute attack of pain in the precordium. His lungs at the time were clear and I felt that there had been a coronary thrombosis. I did not think that there was evidence for atelectasis or pneumonia, and did not believe that a pulmonary condition was playing any part in the symptoms. I did not see him again.

CLINICAL DIAGNOSES

Coronary thrombosis.
Carcinoma of the rectum.

DR. PAUL D. WHITE'S DIAGNOSES

Hypertensive coronary heart disease.
Coronary artery narrowing due to atherosclerosis.
Aortic stenosis.
Pulmonary infarction from embolism?
Cystitis.
Pyelitis?
(Carcinoma of the rectum.)
Paroxysmal auricular fibrillation.
Angina pectoris.
Intermittent claudication.

ANATOMIC DIAGNOSES

(Carcinoma of the rectum.)
Operative wound: Combined abdominoperineal resection of the rectum with colostomy.
Pulmonary embolism, bilateral.
Coronary thromboses with myocardial infarction, old.
Pulmonary tuberculosis, bilateral, active.
Pleuritis, chronic fibrous, bilateral.
Arteriosclerosis, marked coronary and aortic; moderate renal.

Pyelonephritis, slight.
Cystitis, moderate.

Pathologic Discussion

Dr. Mallory: The autopsy showed that Dr. White was correct in predicting that no acute coronary thrombosis would be found. The left coronary artery was completely occluded, however, along with its major branches, but the occlusion was evidently very old. The descending branch of this artery was reduplicated, and was represented by a small obliterated cord-like vessel and a somewhat larger, almost completely closed one. The circumflex branch of the left was also completely closed. The right coronary was narrow but not obliterated. There were three small separate areas of infarction, all of them old and completely scarred. In one of them the scar tissue was rather vascular and it is conceivable it may have developed at the time of the first operation, at any rate it seemed consistent with a duration of about a month. The other scars were evidently of much longer standing. We also found pulmonary emboli. He had an embolus to each of the lower lobes, the one on the right side being evidently much older than the one on the left, so I think perhaps the attack of right chest pain after the operation was due to the embolus.

The immediate cause of death was not entirely certain. He undoubtedly had a bad heart but we found one other totally unsuspected complication that I am sure played a significant rôle. The upper half of each upper lobe was completely consolidated with old tuberculosis and there were fresh foci of tuberculosis throughout both lungs. If Dr. King had had a chance to examine him at an earlier stage, before the operation, I am sure he would have found something more definite. The aortic valve was described as not stenotic but the operator made note that the commissures and the valve margins were thickened and he measured the circumference as only six centimeters, so that in spite of his statement I believe there was a slightly stenotic but not a massively calcified valve.

A Physician: May I ask Dr. White a question as regards postoperative fluids in cardiac patients? What is your opinion of the method of giving them, intravenously or hypodermically?

Dr. White: If, as in this man, patients show easy induction of angina pectoris or if congestive failure is impending, I think extra fluid should be given slowly and not rapidly. I should think subpectoral hypodermoclysis is wiser in such cases than rapid intravenous injection.

CASE 22172

Presentation of Case

A seventy-three year old retired American insurance agent was admitted in coma.

The patient had always lived a vigorous life and had eaten heartily of a well-seasoned diet with only occasional gastrointestinal upsets until one year before entry. At this time he began to have more frequent similar upsets, which assumed the character of gnawing and burning sensations in the epigastrium, rarely occurring under two hours after a meal and usually appearing at or after going to bed. This discomfort was somewhat relieved by further ingestion of food or fluid. Three months ago he consulted a physician who found something wrong with his stools and told him he had an ulcer although no x-ray was taken. He was given iron pills and a powder to take every four hours. He remained in bed for two weeks, adopted a bland diet, and at the end of a month was almost completely relieved of his symptoms. Thereafter he returned to work but had very little success. For several years he had been informed by his employers that his work was becoming poor and that he frequently made mistakes indicative of poor judgment. Following his return to work his attempts to obtain business were unsuccessful. He began to look sallow and his weight, which had been 250 pounds fifteen years before entry and had gradually diminished to 200 one year ago, now became approximately 175 pounds. Six days prior to admission he visited his daughter who stated that he looked weak, pale, and listless. His appetite was poor and, contrary to his previous assertive manner, he was very much subdued. He went to bed and appeared to remain asleep ever since. He was aroused only with difficulty, was rather restless but uncommunicative, and his skin was cold and clammy.

For years he had been told that his blood pressure was consistent with his age, but eight months ago it was said to be 220.

The patient's father and brother had both had "ulcers of the stomach". His mother had had Bright's disease.

Physical examination showed a pale, slightly obese, elderly male in restless coma. The mucous membranes were pallid, the tongue dry, and the breath had no characteristic odor. The pupils were unequal, the right being larger than the left. They reacted poorly to light. The lungs were clear. The heart extended 8 centimeters to the left of the midsternal line and 2 centimeters to the right. The rhythm was totally irregular and no murmurs were audible. The blood pressure was 120/80. The abdomen was large and flaccid. No masses were palpable, nor was there evident local spasm or evidence of tenderness. A rectal examination showed a moderately enlarged prostate but no palpable masses. The examining finger was covered with blood-stained fecal material. A neurologic examination was negative.

The temperature was 100.8°, the pulse 100. The respirations were 30.

Examination of the urine showed a specific gravity of 1.014 and there was no sugar, albumin, or diacetic acid. The sediment was negative. The blood showed a red cell count of 2,820,000, with a hemoglobin of 60 per cent. The white cell count was 19,100, 90 per cent polymorphonuclears. A Hinton test was negative. The nonprotein nitrogen of the blood was 120 milligrams per cent. A fasting blood sugar was 159 milligrams. A lumbar puncture showed clear colorless fluid with an initial pressure of 50 millimeters. The ammonium sulphate test was negative and there were two cells per cubic millimeter. The total protein was 58 milligrams. An electrocardiogram showed auricular fibrillation with moderate left axis deviation. S_2 and S_3 were prominent. There was a deep Q_4 and inverted T_4.

The patient continued comatose and two other urine specimens showed no significant abnormality. On the second day the nonprotein nitrogen of the blood was 153 milligrams. The temperature subsided to 99° but the patient did not regain consciousness. He died on the second hospital day.

Differential Diagnosis

Dr. Allen G. Brailey: I think it is a very interesting case, and with this confusion of signs and symptoms one wishes one could rely on the old diagnosis of "complication of diseases". I think the place to begin is at the end. A man seventy-three years old dies in coma after five days to a week. Most of the causes of fatal coma are pretty easily eliminated. There is no evidence of trauma, for instance, and poisoning seems to be out of it. He has lost blood; he has anemia; the white count raised; but he certainly did not die of blood loss. Then there is a cerebral accident. It seems to me the onset of the coma was too slow for ordinary cerebral accident. The neurologic examination was negative except for minor differences in pupils. The spinal fluid was essentially normal. The total protein was 58. The pressure was 50 millimeters, which is very low. I do not know how to interpret either one of these figures unless they are the result of dehydration in a man who has been asleep for five or six days, and has had very little fluid. At any rate I think it is unlikely that his coma was due to any intercerebral condition. I think diabetes is pretty well ruled out. There is no characteristic odor to the breath. The blood sugar is somewhat raised, but not enough to be significant.

However, this is a very characteristic picture of uremic coma. He does not have convulsions but is restless. He had previous loss of appetite. In fact, going over the past history, for some time he has been under the weather. He has been losing weight in significant fashion for a year and there has been less efficiency at business. Then, also, he is anemic. The anemia may well be bleeding from the intestinal tract which is probably due to colitis or ileitis which is so common with uremia. Of course uremia with a nonprotein nitrogen of 120 scarcely can be due to anything but chronic kidney disease. It occurs with prolonged vomiting in high intestinal obstruction but there is no reason to suppose that is the case here.

There are some things against this picture. If he has chronic kidney disease presumably he has had high blood pressure for some time and other evidence of renal and vascular damage. The record says that eight months ago the pressure was 220 but the one taken in the hospital was normal. The heart is described as small,—so small I think he undoubtedly has an emphysema and the percussion was unreliable. Still it must be admitted there is no evidence of its being enlarged. Then, of course, he had no convulsions. His urine on three examinations was normal. As a matter of fact they were not normal. The only one we have accurately reported is the first, with a specific gravity of 1.014. He is an old man and probably had taken very little fluid for five or six days and he is obviously dehydrated, so that the specific gravity of 1.014 is very significant of inability to concentrate. It is interesting that he has no albumin, no blood and no casts; it is unusual but not unheard of and does not make me change my mind about the diagnosis. I think there are a lot of contradictory statements here but I think he has had kidney disease of long standing and that he has uremia with colitis or ileitis, or both.

What other things do we have to consider? His doctor said he had an ulcer.

But the story of ulcer so far as it goes is not typical. He has had some gnawing, burning epigastric discomfort daily for a year, occurring for the first time in an old man. It is not well relieved by food. To be sure he gets better when he goes to bed with a bland diet and rest, but almost any stomach condition will improve under these conditions. He may have an ulcer, but I do not feel obliged to suppose so. I think his symptoms can be better explained on the basis of uremia. Now as to carcinoma, he is an old man who has lost weight, who has anemia and bloody stools, but again I prefer the diagnosis of uremia with gastrointestinal bleeding.

What is the matter with his heart? He may perfectly well have had coronary accident. We notice the blood pressure is lower than it was said to have been. He has leucocytosis and fever. In fact, coma may conceivably be due to emboli from the left auricle. But the electrocardiogram does not indicate coronary occlusion. I suppose he probably does have some infection to account for fever and leucocytosis, perhaps associated with the colitis, which I think he has. Also he may very well have a small amount of bronchopneumonia.

Dr. Tracy B. Mallory: I might again ask if there are any alternative diagnoses.

Dr. Wyman Richardson: I suspect this man

also has a duodenal ulcer. I should like to inquire about the danger of treating an old man with duodenal ulcer with that large amount of alkali, particularly if he has renal disease, vascular nephritis.

DR. MALLORY: I do not know how to answer that.

DR. WILLIAM B. BREED: I think that one cannot dismiss the diagnosis of carcinoma of the stomach here. It is of only a year's duration and we know of many cases of cancer of the stomach who have over a short period symptoms suggesting ulcer. He has lost a great deal of weight, which is difficult to explain entirely on the basis of kidney disease, and I think we should not lose sight of the possibility of cancer of the stomach.

DR. EARLE M. CHAPMAN: Will Dr. Breed explain the final episode with brain metastases of carcinoma.

DR. BREED: Certainly not.

CLINICAL DIAGNOSES

Chronic nephritis.
Uremia.
Gastrointestinal bleeding.

DR. ALLEN G. BRAILEY'S DIAGNOSES

Chronic nephritis.
Uremia.
Gastrointestinal bleeding, probably from the colitis of uremia.

ANATOMIC DIAGNOSES

(Uremia.)
Nephritis, chronic vascular.
Duodenal ulcer, chronic, without perforation.
Cardiac hypertrophy, hypertensive.
Arteriosclerosis, moderate aortic and coronary.
Cholelithiasis.
Choledocholithiasis.
Pulmonary edema and congestion, bilateral.
Cystitis, acute localized.
Dilatation of the esophageal hiatus.
Operative scar: Amputation of the distal phalanx of the right first finger.

PATHOLOGIC DISCUSSION

DR. MALLORY: This man showed a very large penetrating ulcer on the posterior duodenal wall which had burrowed into the pancreas. He showed a greatly hypertrophied heart — Dr. Brailey was wise in questioning the apparent facts in the record. It weighed 650 grams, which is double the normal. His kidneys weighed 325 grams, which is the lower limit of normal for a man, and they showed a little scarring on the surface but the cortex measured 5 millimeters, which is pretty good for anyone of seventy. Microscopically, they showed very definite changes in the arteries and a few changes in the glomeruli which are consistent with long-standing hypertension, but they were not the kidneys that one would expect to find in a patient in uremia. He still had rather too much gross kidney substance for death from vascular nephritis and they also failed to show one other thing which is probably of some importance. One of the first clinical signs of insufficient renal function, whatever the origin, is lack of tubular function; in other words, lack of concentration. Drs. Wilson and Kimmelstiel in going over 100 cases of vascular nephritis at the Boston City Hospital, found that the best correlation they could work out between the anatomic findings in the kidney and the functional evidence of renal failure depended upon the observation of dilatation of the tubules. Practically no cases showed real evidence of renal failure unless the tubules were fairly markedly dilated and hyperplastic. This man showed absolutely no dilatation of the tubules, so I should say that they were not the kidneys that I would expect to have produced uremia. Recently Dr. Castleman has run across in Scandinavian publications several reports of uremic states following hemorrhage from the gastrointestinal tract. A man named Meyler* has reported six cases of impending or established uremia with a nonprotein nitrogen over 100 in patients with severe gastrointestinal hemorrhage. Some of these cases came to autopsy and were found to have relatively normal kidneys. Some recovered following appropriate therapy and the signs of renal failure disappeared.

I am inclined to raise the question concerning this case today whether we are not dealing with a synergistic effect of mildly damaged kidneys plus at least a moderate amount, perhaps —we are not sure—a very considerable amount of gastrointestinal hemorrhage whether the hemorrhage from the gastrointestinal tract may not actually have played a rôle in producing the uremia. It is not an entirely satisfactory case because we did not have permission to examine the head.

A PHYSICIAN: Such an effect would be explained on the basis of damaged circulation of the kidney?

DR. MALLORY: Yes; the mechanism is rather uncertain and, although there are a considerable number of reports of the condition of nitrogen retention associated with hemorrhage, the various authors who have reported the cases differ widely in their theories of the pathogenesis. On the whole they think dehydration is most important and that anemia can play some rôle but a relatively unimportant one.

DR. GEORGE W. HOLMES: I have seen with Dr. C. M. Jones two patients who had duodenal ulcers and who illustrate what Dr. Richardson has said. Each of them had some kidney damage and as soon as they were placed upon an alkaline diet their condition became more serious.

*Meyler, L. (Gronigen, Holland): Post-haemorrhagic uraemia. Acta med. Scandinav. 87:313, 1935.

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SERUM TREATMENT OF LOBAR PNEUMONIA

IMPROVEMENTS in the concentration of antipneumococcic serum, chiefly those suggested by Felton, have resulted in establishing the value of serum in the treatment of certain cases of lobar pneumonia. With accurate diagnosis, with proper selection of cases and with adequate doses of potent serum there seems to be little or no doubt but that the mortality in over 50 per cent of all cases of lobar pneumonia can be nearly cut in half. The importance of serum therapy as a life-saving measure is forcefully brought out by Heffron[1] who states that over 18,000 lives in the United States might be saved annually by this method of treatment. This figure is based on an estimated annual total of approximately 128,000 cases of lobar pneumonia and 4,000 cases of bronchopneumonia, all due to Types I and II pneumococci.

Until quite recently this method of treatment has been considered possible only in the larger medical centers and there only in the larger hospitals. The Massachusetts Pneumonia Study, financed by the Commonwealth Fund and carried out from 1931 through 1935 by the Massachusetts Department of Public Health, has shown that equally successful results can be obtained in smaller communities and by physicians without hospital connections in the larger centers, provided typing facilities are available and provided the treatment is properly carried out. Based on these findings, the Massachusetts Department of Public Health has recently announced its willingness to supply all physicians in the Commonwealth with antipneumococcic serum, subject to certain reasonable restrictions. Similar action has been taken by the Departments of Health of Connecticut, New York State and Detroit. A valuable, but expensive, therapeutic agent is thus made available and the responsibility for its judicious use falls entirely upon the practicing physician.

As an aid in properly discharging this responsibility a small book, "Lobar Pneumonia and Serum Therapy", recently published by the Commonwealth Fund, should prove invaluable. The authors, Dr. Frederick T. Lord and Dr. Roderick Heffron, are well qualified—the former being a member of the Massachusetts Advisory Committee on Pneumonia and the latter Field Director of the Massachusetts Pneumonia Study. The important questions of diagnosis, selection of cases, precautions and methods of treatment are simply and briefly, but adequately, discussed. The success of serum therapy in lobar pneumonia depends, in a large measure, upon its intelligent use. The text of this handbook is based, chiefly, on data from the Massachusetts Pneumonia Study, a highly successful five-year experiment, and the book itself cannot be recommended too highly to those who aim to make the best use of a most important addition to their armamentarium.

REFERENCE

1. Letter from Dr. Roderick Heffron. New Eng. J. Med. 214: 222 (Jan. 30) 1936.

PROBLEMS OF THE FLOOD

THE devastating floods which inundated parts of fourteen states in March gave rise, naturally, to a considerable degree of alarm concerning the hazards to health which might follow in their wake. Apprehensions were expressed by many individuals, even those living remote from the flooded districts, of a menace to health in the subsiding waters; a rush for typhoid vaccination was experienced and many families and even physicians prescribed the consumption of raw fruits and vegetables, regardless of their source.

It is natural that the specter of typhoid fever should arise when vast areas of settled country are covered by possibly polluted waters. Undoubtedly countless cesspools and

privies have been irrigated by the swollen rivers. Two factors of safety, however, lie in the incalculable dilution which the contents of contaminated foci have experienced, and the fact that in most sections, at least, municipal water supplies have remained unpolluted. As a state health official recently pointed out, all public water supplies in Massachusetts were examined following the floods, the only one showing any suspicion of contamination coming from outside the flood areas.

According to the statement of this same official, the Massachusetts floods presented a problem of relief rather than of public health administration, although the Department of Public Health discharged its obvious duties immediately, efficiently and conservatively. Actually exposure and destitution provided the immediate health hazards, pneumonia constituted a graver risk than did typhoid fever, and the pressing need at the time was for food, shelter and clothing.

Continuing problems are being met as rapidly as possible. The débris is being cleared away and homes are being renovated. Roads are being opened and bridges replaced in order that life may resume its normal course. In some sections arable lands have been stripped of their top soil and left barren; in others the fields have been enriched by the deposit of silt left upon them. As Commissioner Chadwick has indicated, however, these newly dressed fields must be cultivated and sown as soon as possible, lest the drying winds of summer again scatter the soil abroad before the roots of a new crop have anchored it to its place.

TUBERCULOSIS OUTBREAK IN AN ACCREDITED HERD

An unexplained, extensive outbreak of tuberculosis in the Government's herd of dairy cattle at Beltsville, Maryland, is being investigated by the United States Department of Agriculture, according to a recent release. In this outbreak eighty-two positive reactors and eleven suspicious animals were discovered in a previously clean herd of 378 animals. Of thirty-one reactors and suspects slaughtered at the time of this report, twenty-five showed tuberculous lesions, practically all of these being in cervical and thoracic lymph nodes.

This herd has been for eighteen years in an accredited status. In April, 1935, on a periodic test, one animal was regarded as suspicious. A ninety-day retest in July showed three reactors; a further retest in October disclosed one infected animal. Reactors appeared in seven of eight buildings in January, only the bull barn escaping infection. For seven years the herd has been maintained almost entirely by replacements raised on the farm, the few cattle from outside sources having come from accredited herds.

The theory of accreditation, by herd or area eradication, is eventually to establish and maintain a tuberculosis-free cattle population. If bovine tuberculosis could be completely wiped out, there would, in theory, be no source for further infection and the millennium would be reached, in this respect at least. Complete eradication, however, is obviously difficult of achievement and similar outbreaks of unknown source, in accredited herds, have not been unknown. An accredited herd or an accredited area is a laudable ambition, but the goal once reached cannot be accepted with complacence, for continued enjoyment of it is at the cost of eternal vigilance.

A tuberculosis-free herd may become, in time, a highly susceptible herd, and frequent retests with ideal conditions for isolation and segregation are necessary for its maintenance.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Haggart, G. E. A.B., M.D. Harvard University Medical School 1919. F.A.C.S. Orthopedic Surgeon, Lahey Clinic; New England Deaconess Hospital, and New England Baptist Hospital. Address: 605 Commonwealth Avenue, Boston, Mass. Associated with him is

Peelen, Matthew. M.D. Rush Medical College 1931. Orthopedic Surgeon, Lahey Clinic, 1933-34. Address: Kalamazoo, Mich. Their subject is Nonunion in Shaft Fractures of the Humerus. Page 815.

Richter, Arthur B. A.B., M.D. Indiana University School of Medicine 1931. Previous Internships at Indiana University Hospitals, Peter Bent Brigham Hospital, and Cleveland City Hospital. Assistant Resident Physician, Peter Bent Brigham Hospital, 1935. Now practicing in Flora, Ind. Address: Flora, Ind. Associated with him is

O'Hare, James P. A.B., M.D. Harvard University Medical School 1911. Assistant Professor of Medicine, Harvard University Medical School. Senior Associate in Medicine, Peter Bent Brigham Hospital. Address: 520 Commonwealth Avenue, Boston, Mass. Their subject is The Heart in Chronic Glomerular Nephritis. Page 824.

Wheeler, Philip H. A.B., M.D. Harvard University Medical School 1930. Assistant Surgeon, Brattleboro Memorial Hospital. Junior Consulting Surgeon, The Northfield (Mass.) Schools. His subject is Enormous Benign Gastric Ulceration Caused by Multiple Foreign

Bodies. Page 830. Address: 4 Elliot Street, Brattleboro, Vt.

BAKST, HENRY J. Ph.B., M.D. Harvard University Medical School 1931. Junior Visiting Physician, Boston City Hospital. Assistant in Medicine, Boston University School of Medicine. Address: 482 Beacon Street, Boston, Mass. Associated with him are

WETHERBEE, WINTHROP, JR. A.B., M.D. Columbia University College of Physicians and Surgeons 1931. Junior Visiting Physician, Boston City Hospital. Assistant in Medicine, Boston University School of Medicine. Address: 482 Beacon Street, Boston, Mass. And

FOLEY, JOHN A. A.B., M.D. Harvard University Medical School 1915. Clinical Professor of Medicine, Boston University School of Medicine. Visiting Physician and Physician-in-Chief, Fifth Medical Service, Boston City Hospital. Address: 464 Commonwealth Avenue, Boston, Mass. Their subject is Orthostatic Albuminuria in Homologous Twins. Page 832.

LOW, MERRITT B. A.B., M.D. Harvard University Medical School 1933. Formerly, Surgical House Officer, Children's Hospital, Boston. Medical House Officer, Children's Hospital of Philadelphia. Now, Surgical Resident, Children's Hospital, Boston. His subject is Anorexia in Children. Page 834. Address: Children's Hospital, Boston, Mass.

WHITNEY, C. F. B.S., M.S., M.D. University of Vermont College of Medicine 1903. Director, Laboratory of Hygiene, Department of Public Health, Vermont. Professor of Physiological Chemistry and Toxicology, University of Vermont. His subject is Chemistry in Relation to the Practice of Medicine. Page 837. Address: Burlington, Vermont.

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TREATMENT OF CHRONIC HEART DISEASE COMPLICATING PREGNANCY

As is to be expected, treatment of cardiacs in pregnancy is based chiefly on (1) preventing heart failure; (2) recognizing it early if it occurs; and (3) treating it adequately.

Though the great majority of failures occur in the last trimester of pregnancy, the rule that *any cardiac may develop heart failure at any time during pregnancy or puerperium* must be appreciated. Clearly every human being who has heart disease should be under medical supervision, and part of the supervision of married women of child-bearing age who have heart disease should be specific instruction to report at once if pregnancy is suspected. A suitable régime should be instituted immediately.

The management of cardiacs is based, in general, on a study of unsuccessful cases. The commonest cause of failure is accumulating fatigue. Therefore, a schedule of regular hours of rest at night and for one or more periods during the day is essential. Patients should be instructed never to break these fundamental rules under any circumstances. The nature and duration of house or other work and of walking; the number of flights of stairs each day; should be carefully discussed with the patient and regulated. Clearly no set rules can be made that are appropriate to all women. In general, I have never seen any real harm come from restriction of activity and I have seen much harm from failure to control activity adequately. Many failures could be averted if patients were carefully instructed on these simple matters and persuaded to follow their instructions. The commonest causes for breaking these rules and allowing fatigue to occur are the following: sickness in the patient's family, journeys, shopping trips, moving the household either to or from a summer home or to another home.

Next to balancing rest and exertion, diet is probably the most important single factor. Proper diet should cover the following points: (1) weight control. Weight reduction is of immense benefit to obese cardiacs. Patients who are not obese should not be allowed to gain more than the standard amount during pregnancy. Meals should be regular and at no time should a patient be allowed to gorge. (2) *Fluid intake* should be controlled. Excessive fluids particularly must be avoided where heart failure is present or feared. (3) Adequate nutrition and (4) control of anemia. Fruits, meats, vegetables and milk must be taken to aid in maintaining a satisfactory high red blood cell count and hemoglobin. Anemia seems to be an especially severe burden on cardiac patients in pregnancy, and it should be treated vigorously by drugs if diet control is insufficient. There is no reason why the modern accepted requirements for nutrition of the mother cannot be made to fit the requirements of the cardiac patient.

Oral sepsis may be a heavy burden and should be searched for and treated reasonably if found.

The rôle of infection in producing heart failure in pregnancy is as follows: the typical rheumatic heart disease patient in pregnancy has acquired a satisfactory immunity to recur-

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

rence of the active rheumatic disease. This is a fortunate and not thoroughly appreciated fact. We do not need to dread a flare-up of rheumatic carditis. It is an extremely rare occurrence during pregnancy. Intercurrent infections—tonsillitis, grippe and the like—occur more often than not at some time during a pregnancy. They have a certain danger. It is remarkable how well cardiac patients tolerate these illnesses if they go to bed at the onset of symptoms and stay there until the infection is thoroughly over, then slowly increase their activities, taking a week or two to return to the level of physical exercise allowed before the infection. They seldom develop failure. If these precautions are neglected, the average upper respiratory infection may readily cause heart failure in a cardiac during pregnancy.

Patients with cardiac disorders must report for examination at least once a week. The main object of these visits is to detect early signs of failure. Though refinements of observation, for example changes in the movements of the chest wall with respiration, and careful vital capacity determinations may in some cases warn of a threatening failure, the earliest reliable signs of heart failure are persistent râles at the lung bases posteriorly. If a cardiac develops these râles during pregnancy, she should be considered to be in failure.

Failure means that the patient has suffered serious damage. The usual treatment for heart failure should be started and *the patient should be in bed, or in bed or chair, until delivered*, no matter how promptly and completely the signs of failure may clear with treatment. Failure in any pregnancy also means that the patient faces approximately a death rate of 25 per cent if she tries to go through another pregnancy.

An immense amount of ingenuity can profitably be used in selecting type and time of delivery, in selecting the form of anesthesia and in its administration, in careful preparation and nursing of the patient through labor, delivery and recovery from operation. No set rules can or should be followed. Fortunately, granted skillful delivery and aftercare, few cardiacs fail for the first time during or following delivery. The emphasis of the treatment of heart disease during pregnancy should be on careful early diagnosis of the heart disease and relentless control of the cardiac patient throughout the whole of pregnancy, delivery and puerperium. I have deliberately accented these apparently simple details of prenatal care of the cardiacs since these are by all odds the most important part of the problem.

THIRD ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning April 26:

Berkshire

Thursday, April 30, at 4:30 P.M. at the House of Mercy Hospital, Pittsfield. Subject: Kidney and Bladder Diseases A (Surgical)—Hematuria; Its Significance in Surgical Diseases of Kidney and Bladder. Instructor: F. H. Colby. Melvin H. Walker, Jr., Chairman.

Bristol North

Wednesday, April 29, at 7:30 P.M., at the Morton Hospital, Taunton. Subject: Pediatrics (Medical)—The Neonatal State. Instructor: W. R. Sisson. Arthur R. Crandell, Chairman.

Bristol South (New Bedford Section)

Friday, May 1, at 4:00 P.M., at the St. Luke's Hospital, New Bedford. Subject: Diseases of the Liver — Hepatitis and Painless Jaundice. Problems in Diagnosis and Treatment. Instructor: C. Frothingham. Harold E. Perry, Chairman.

Franklin

Wednesday, April 29, at 8:00 P.M., at the Franklin County Public Hospital, Greenfield. Subject: Dermatology—Ten Common Skin Diseases—Diagnosis and Treatment. (1) Impetigo Contagiosa, (2) Scabies, (3) Acne Vulgaris, (4) Psoriasis and Seborrhoeic Dermatitis, (5) Epidermophytosis, (6) Herpes Simplex and Zoster, (7) Eczema, (8) Erythema Multiforme, (9) Verruca Vulgaris and (10) Dermatitis Medicamentosa and Dermatitis Venenata. Instructor: J. H. Blaisdell. Halbert G. Stetson, Chairman.

Middlesex East

Wednesday, April 29, at 4:00 P.M., at the Melrose Hospital, Melrose. Subject: Kidney and Bladder Diseases A (Surgical)—Hematuria; Its Significance in Surgical Diseases of Kidney and Bladder. Instructor: G. C. Prather. Joseph H. Fay, Chairman.

Middlesex North

Friday, May 1, at 7:30 P.M., at the Lowell General Hospital, Lowell. Subject: Cancer of Breast and Uterus. Instructor: J. V. Meigs. Leonard C. Dursthoff, Chairman.

Norfolk

Friday, May 1, at 8:30 P.M., at the Norwood Hospital, Norwood. Subject: Lung Diseases—(a) Significance of Symptoms and Signs in Chronic Lung Diseases; Tuberculosis, Bronchiectasis, etc. (b) The Value of Surgery in Above Disease Problems. Instructor: S. H. Proger and R. H. Sweet. H. B. C. Riemer, Chairman.

Worcester (Milford Section)

Wednesday, April 29, at 8:30 P.M., at the Milford Hospital, Milford. Subject: Arthritis—(a) Medical Care of Patient in the Home.

(b) Orthopedic Treatment in Hospital and Aids in Home Treatment. Instructors: R. T. Monroe and W. T. Green. Joseph I. Ashkins, Sub-Chairman.

APPLICATION FOR MEMBERSHIP IN THE ESSEX NORTH DISTRICT MEDICAL SOCIETY

New England Journal of Medicine,

According to the resolution adopted at the last Council meeting, I am sending you the names and addresses of an applicant for Fellowship and five sponsors which are to be published three weeks prior to the Censors' meeting.

Applicant — Dr. Lawrence Murphy, 216 High Street, Newburyport, Mass.

Sponsors—Dr. Frank W. Snow, 24 Essex Street, Newburyport; Dr. R. L. Toppan, 148 High Street, Newburyport; Dr. C. F. A. Hall, 210 High Street, Newburyport; Dr. L. C. Peirce, 279 High Street, Newburyport; Dr. R. C. Hurd, 244 High Street, Newburyport.

In compliance with the By-Laws of the Society (Chap. I, Sec. 1; Chap. V, Sec. 1; and Chap. VII, Sec. 5) the application and letters of the sponsors have been sent to Dr. Fitz, Chairman of the Committee on Medical Education and Diplomas.

E. S. BAGNALL, M.D.

April 14, 1936

MISCELLANY

$10,000 PRIZE FOR RELIEF OR CURE OF DISEASES OF REPRODUCTIVE ORGANS

A prize of more than $10,000 will be awarded in 1940 and every seven years thereafter by the American Academy of Arts and Sciences in Boston for "outstanding work with reference to the alleviation or cure of diseases affecting the human genital organs". The award is to be known as the Francis Amory Septennial Prize, since it is made possible by a fund established by the will of the late Francis Amory of Beverly, Massachusetts. In case there is work of a quality to warrant it, the first award will be made in 1940. It rests solely within the discretion of the Academy whether an award shall be made at the end of any given seven-year period, and also whether on any occasion the prize shall be awarded to more than a single individual.

While there will be no formal nominations, and no formal essays or treatises will be required, the committee invites suggestions, which should be made to the Amory Fund Committee, care of the American Academy of Arts and Sciences, 28 Newbury Street, Boston.—*The Diplomate, April 1936, page 136.*

"DR. JIM," HEALER FOR 1,000,000 ZULUS, RETURNS FROM THIRTY-FIVE YEARS IN SOUTH AFRICA

"Dr. Jim," physician for over thirty-five years in Durban, South Africa, arrived in Boston April 14.

In Africa, where he founded the first hospital for Zulus in 1899, that is his title. His hospital is the "House That Jim Built," and his wife, a trained nurse, is "the Princess".

His New England friends who were skeptical when he left here as a young missionary doctor, knew him as Dr. James B. McCord of Oakham, Mass.

"There is only about one doctor to every million people in Africa," Dr. McCord informed us.

To meet this great medical need, Dr. McCord is training the black to care for his own, as well as relieving sickness and suffering in his hospital.

Last year, after a seventeen-year struggle, Dr. McCord induced the South African Government to put through a plan whereby young Zulus will receive medical education in the South African Native Colleges.—*Boston Transcript, April 14, 1936.*

AMERICAN COLLEGE OF PHYSICIANS

At the recent annual meeting of the American College of Physicians, held in Detroit, the following members assumed office or were elected for the year 1936-1937:

President, Ernest B. Bradley, Lexington, Ky.

President-Elect, James H. Means, Boston.

First Vice-President, O. H. Perry Pepper, Philadelphia.

Second Vice-President, David P. Barr, St. Louis.

Third Vice-President, Walter L. Bierring, Des Moines.

Secretary-General, William Gerry Morgan, Washington, D. C.

Executive Secretary, E. R. Loveland, Philadelphia.—*The Diplomate, April 1936, page 134.*

ANNUAL JOINT MEETING OF AMERICAN ASSOCIATION OF MILK COMMISSIONS AND CERTIFIED MILK PRODUCERS

Immediately preceding the eighty-seventh annual meeting of the American Medical Association, the American Association of Medical Milk Commissions will hold its annual joint meeting with the Certified Milk Producers' Association of America, May 11-12, at the Hotel Baltimore, Kansas City, Mo.

In addition to reports from the eighty-six Medical Milk Commissioners in the United States, Hawaii and Canada, addresses will be delivered by a number of prominent physicians and scientists. These will include Dr. Milton J. Rosenau, formerly head of the Department of Preventive Medicine and Hygiene at the Harvard Medical School, President of the American Association of Medical Milk Commissions; Dr. Boyd S. Gardner of the Mayo Clinic, Rochester, Minn.; Dr. Paul J. Zentay, Secretary, St. Louis Pure Milk Commission; Dr. Harold L. Barnes, Chairman, King's County Medical Milk Commission, Brooklyn, N. Y.; Dr. Oscar Reiss, Secretary, Los Angeles County Medical Milk Commission; Dr. Edwin T. Wyman, Boston Medical Milk Com-

mission, and Dr. Hugh L. Dwyer, Medical Milk Commission, Jackson County Medical Society, Kansas City, Mo.

WHY TRY TO PERSUADE THE PUBLIC?

Today medicine is where theology was when Gutenberg invented printing.

The Bible, which was the special and sacrosanct possession of a small, highly specialized group, the clerics, soon became, after the invention of printing, a book about which anybody could have an opinion—almost everybody did have an opinion—and the result of the clash of beliefs in extreme instances resulted in the overthrow of governments and in wars. By virtue of the Bible becoming accessible to the public, civilization was profoundly changed.

We live today in another age of change; change which is affecting medicine along with everything else. The loafer on the street corner considers himself competent to express an opinion on the gold standard. What is still worse, competent men in one field consider themselves qualified to express opinions in another field, in which they are not competent.

Thousands of medical men, trained in our medical schools, are occupied in rendering a partial and limited knowledge of medicine widely available to the public, as employees of official and voluntary health organizations, or as popular writers on medical subjects. True, they are imparting only a smattering. Their work is open to the criticism that it indirectly encourages self-diagnosis and self-medication. The increasing growth of this movement cannot be stopped by condemning it, if indeed it should be condemned. A little learning is not a dangerous thing to a man who knows it to be little. If organized medicine co-operates and itself becomes vocal, we will not be overwhelmed in the sea of created opinion which, if it be not altogether opposed to the purposes of organized medicine, is, in substance, indifferent to them.

Justice Oliver Wendell Holmes said: The best test of truth is the power of thought to get itself accepted in the open market.—*Public Relations Bureau, New York State Medical Society.*

THE CONTROVERSY OVER WHETHER ALEXIS ST. MARTIN EVER VISITED ST. LOUIS

For a considerable time the story that Alexis St. Martin visited St. Louis has been disputed.

In the *Bulletin of the Medical Library Association* for February, 1936, there is a copy of an editorial (St. Louis Med. & Surg. J. [July] 1856) found by Miss Harriette Werthmuller, Assistant Librarian of the Library of the St. Louis Medical Society, which establishes the fact that Alexis St. Martin did visit St. Louis in charge of Dr. Bunting.

The text of the editorial is as follows:

ALEXIS ST. MARTIN

Dr. Bunting, late of the British army in Canada, recently visited our city, having in charge St. Martin, so well known in this community, and by physiologists everywhere, as the subject on which our late distinguished fellow-citizen, Dr. Beaumont, performed his celebrated experiments on the physiology of digestion. While here Dr. B. exhibited this unique specimen in the amphitheatre of the St. Louis Medical College, and performed a few so-called experiments before a large number of physicians and other scientific gentlemen of the city, at the close of which a collection was taken up for the benefit of his patient. It is now some thirty-five or six years since St. Martin received a gunshot wound in his left side, which has left a large fistulous opening into his stomach, through which the rare opportunity is afforded of examining the contents of that organ during the various stages of digestion. At present, he is 52 years old, is in good health, and in the full and vigorous possession of all his functions, seeming to suffer but little inconvenience from his wound. When the bandage is removed and the side exposed, the inner coat of the stomach slightly protrudes, presenting a red, almost scarlet, appearance.

It is understood that Dr. Bunting and his attache are on their way to Europe, where it is to be hoped the case will fall into the hands of some of the eminent physiological observers of the old world who are capable of using it for the advancement of science. Such a case has never before occurred, and may not occur again, yet it is greatly to be regretted that as yet but comparatively little practical good has resulted to physiological science from it. In the exhibition which we witnessed, it was evident that the object was more to realize money than to promote science, but we hope that hereafter a different course will be pursued, and that the profession will yet have the benefit of all the light that this rare case is capable of shedding on the interesting subject of digestion. We therefore earnestly desire to hear from St. Martin again.

From this it can be seen that he came about three years after William Beaumont's death which occurred on April 25, 1853.

CHILD HEALTH DAY

On April 13, President Roosevelt proclaimed May 1 as Child Health Day. The text of the proclamation is:

Whereas the Congress by joint resolution of May 18, 1928 (45 stat., 617) has authorized and requested the President of the United States to proclaim annually May 1 as Child Health Day; and

Whereas the health and security of its children are essential to the well-being of the nation; and

Whereas it is advisable this year as we launch the social security program to encourage by every possible means the development of plans to pro-

mote maternal and child health, and to extend child welfare services:

Now, therefore, I, Franklin D. Roosevelt, President of the United States of America, do hereby proclaim and designate the first day of May of this year as Child Health Day, and do urge all agencies, public and private, concerned with the health and welfare of children, on this day to study the plans for Federal, State, and local co-operation in promoting the health and security of children, to note the extent to which those plans have so far been put into effect, and to make arrangements for carrying their benefits to the children in every county in the United States.

BOVINE TUBERCULOSIS IN CONNECTICUT

The U. S. Department of Agriculture has added Connecticut to its list of states practically free of bovine tuberculosis. This brings the number of states in the modified accredited area to thirty-nine.

Tuberculosis-eradication work among the cattle in Connecticut was begun more than a quarter century ago and has been in progress in co-operation with the federal government since 1918. Recently New London, New Haven, and Fairfield counties were added to the official list of modified accredited counties in that state. Official tuberculin testing of cattle in other counties had been completed previously to an extent whereby these counties could be made modified accredited areas.

Co-operation of the livestock owners with state and federal officials made it possible to reduce the incidence of the disease, which formerly was rather widely prevalent. Before an area can be designated as modified accredited, the degree of infection among the cattle must be less than 0.5 per cent as shown by official tests.

When a state becomes a modified accredited area, the most difficult part of the eradication work has been completed. However, it is necessary to prevent the disease from getting new footholds, officials explain. This is done by retesting the formerly infected herds at regular intervals and removing any diseased cattle found.—*Bulletin, U. S. Department of Agriculture.*

CORRESPONDENCE

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Illinois,
April 1, 1936.

Managing Editor, *The New England Journal of Medicine:*

In addition to the articles enumerated in our letter of March 4 the following have been accepted:

Abbott Laboratories
Ophthalmic Ointment Butesin Picrate 1% and Butesin 1%

Cheplin Biological Laboratories, Inc.
Cheplin's Sodium Cacodylate 0.05 Gm. (¾ grain), 1 cc.
Cheplin's Sodium Cacodylate 0.1 Gm. (1½ grains), 1 cc.
Cheplin's Sodium Cacodylate 0.2 Gm. (3 grains), 1 cc.
Cheplin's Sodium Cacodylate 0.3 Gm. (5 grains), 1 cc.
Cheplin's Sodium Cacodylate 0.5 Gm. (7½ grains), 1 cc.
Cheplin's Sodium Cacodylate 1.0 Gm. (15½ grains), 2 cc.

Lakeside Laboratories, Inc.
Ampoule Solution Sodium Cacodylate 3 grains (0.195 Gm.) 1 cc.

Eli Lilly and Company
Tablets Amytal, ¾ grain
Ophthalmic Ointment Metycaine 4 per cent

National Drug Company
Antipneumococcic Serum-Felton — Type I (Refined and Concentrated)
Antipneumococcic Serum-Felton—Types I and II (Refined and Concentrated)

Parke, Davis & Co.
Kapseals of Ortal Sodium Phenacetin

U. S. Standard Products Co.
Scarlet Fever Streptococcus Toxin for Immunization
Ampule Compound Solution of Calcium Gluconate 10%, 10 cc.

PAUL NICHOLAS LEECH, *Secretary,*
Council on Pharmacy and Chemistry.

RECENT DEATHS

HILL—THOMAS CHITTENDEN HILL, M.D., formerly of Gloucester, Massachusetts, with an office at 270 Commonwealth Avenue, Boston, died at his winter home at Vero Beach, Florida, April 11, 1936.

Dr. Hill was born in Charlotte, Vermont, in 1871, and graduated from Vermont University and the University of Vermont College of Medicine. He subsequently studied at St. Mark's Hospital, London, England. Dr. Hill was a Fellow of the Massachusetts Medical Society, the American Medical Association, and the American College of Surgeons. His specialty was proctology. He was the author of the "Manual of Proctology" which passed through three editions and was translated into foreign languages. By reason of his standing in the specialty, he was elected to the presidency of the American Proctological Society and to the teaching staff of the Harvard Medical School.

Dr. Hill is survived by his widow, the former Mrs. Marion Whitin Brewer; two stepsons, Cyrus and Whitin Brewer, and two brothers, Monroe and Martin Hill, of Charlotte, Vermont.

McGRAW—ANDREW JAMES MCGRAW, M.D., of 93 Washington Street, Taunton, Mass., died April 20, 1936, after a long illness.

Dr. McGraw was a native of Fall River, the son of Hugh McGraw. He graduated from the College of Physicians and Surgeons of Baltimore in 1906 and settled in Taunton soon after. He joined the Massachusetts Medical Society in 1912 and was also a Fellow of the American Medical Association.

Dr. McGraw became interested in civic affairs, was a member of the School Committee for many years and served the City of Taunton as mayor for eight years. During the World War he trained at Plattsburg and was assigned to the 30th Ohio Infantry with which he went overseas, and was retired with the rank of major.

His widow, Mrs. Anna McGraw; two sons, John and Andrew, of Taunton; two sisters, Mrs. Rose McDonald and Mrs. Mary Perry, of Westford, and two brothers, Edward McGraw, of Westford, and John McGraw, of Ohio, survive him.

BULKELEY—FRANK S. BULKELEY, M.D., of Ayer, Massachusetts, died at the Ayer Hospital, April 19, 1936, after an extended illness. Dr. Bulkeley was born in Ayer in 1879, the son of Joseph W. and Serena (Taft) Bulkeley. After studying at Phillips Andover Academy, he entered the Harvard Medical School, graduating therefrom in 1902 and immediately afterward settled in Ayer. He had served as medical examiner of the North Middlesex District for twenty-four years.

Dr. Bulkeley joined the Massachusetts Medical Society in 1902 and terminated his membership in 1918.

KELLY—JOHN S. KELLY, M.D., of Quincy, with an office on Harvard Avenue, Allston, died as the result of an automobile accident, April 20, 1936.

Dr. Kelly was born in 1887 and graduated from the Middlesex College of Medicine and Surgery in 1921.

His widow and three children survive him.

NOTICES

THE HENRY JACKSON LECTURES OFFERED BY THE NEW ENGLAND HEART ASSOCIATION

These lectures will be given by Tinsley R. Harrison, M.D., Associate Professor of Medicine, Vanderbilt University School of Medicine, at 4:45 P.M. on Thursday, April 30, and Friday, May 1, at the Boston Medical Library (John Ware Hall).

Subjects:

1. The Pathogenesis of Circulatory Failure.
2. The Principles of Therapy in Patients with Congestive Heart Failure.

Physicians and students of medicine are cordially invited to attend.

The annual business meeting of the New England Heart Association will precede the lecture on April 30.

MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, April 30, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students.

This will be the last clinic until the first Thursday in October.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian.

REPORTS AND NOTICES OF MEETINGS

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

A meeting of the Middlesex South District Medical Society was held on March 18, 1936 at St. Elizabeth's Hospital. The attendance was unusually large, 125 members being present. The meeting was called to order by Dr. Remick, the president. He introduced Father Brennan who welcomed the members present, expressing his regret about the inclement weather and the pleasure of the hospital in offering its facilities to the District Society.

Seven very interesting papers were presented by various members of the staff. Short summaries follow:

ALEXANDER A. LEVI, M.D.
Secretary, Middlesex South District.

Dr. George F. Keenan, Surgeon-in-Chief, East Surgical Service, presented a Case of Abdominal Pregnancy of Ovarian Origin. The condition was encountered as an unexpected complication in hysterectomy for uterine fibroids.

The patient presented no signs or symptoms of pregnancy. The Aschheim-Zondek test was negative. The classification of all types of extra-uterine pregnancy was briefly reviewed.

Specimens exhibited were a fibroid uterus, right ovary with placental attachment and invasion, and a four months' normal fetus.

Dr. Edward J. O'Brien showed a most interesting case of Bilateral Pyonephrosis with a massive calculus in the left kidney with the lantern slide demonstration of the same.

It was the case of a girl, twenty-six years of age, who had a giant calculus of the left kidney with loss of function on this side, complicated by a massive pyonephrosis of the right kidney which filled the entire right side of the abdominal cavity, to the extent that a gastrointestinal series demonstrated that the entire ascending colon and transverse colon were displaced to the left side of the abdomen. The stone was removed, followed by

the return of function to the left kidney. The right kidney was later operated on and drained with a view to restoring some of its function.

Dr. John E. Burns gave a short talk on Bronchoscopy as an Aid in Diagnosis with Particular Reference to Pneumonography. He showed several lantern slides illustrating the value of lipiodol in mapping out lung pathology.

Dr. John G. Downing spoke of the present day status of Industrial Dermatitis, citing instances of various legal conditions in the different states. He also spoke of the legal problems encountered in some cases here in Massachusetts, and discussed the various dermatoses as defined in law, with reference to a classification which he has compiled from personal cases and the literature, which is as follows:

1. Mechanical or physical agents, i.e, abrasives, heat, cold, actinic rays, friction.
2. Flowering plants and their products—resins, lacquers, wood dusts.
3. Vital agencies:
   a. Bacteria, yeasts, molds.
   b. Members of the animal kingdom—mites, etc.
4. Chemical agents:
   a. Inorganic compounds—acids, bases, salts.
   b. Hydrocarbons and crude coal tar products —oils, tar, pitch, etc.
   c. Other organic compounds.

He then showed lantern slides depicting the patch test (which he explained), and also pictures of characteristic types of Industrial Eruptions.

Dr. Edward M. Hodgkins stated that he was in the habit of regarding the space through which either direct or indirect inguinal hernia occurred as triangular; the boundaries being, the internal oblique and transversalis muscles above the lateral border of the rectus muscle and its fused sheath medially and the spine of the pubis and inguinal ligament below. The triangle becomes dilated by atrophy and retraction medially of the internal oblique and transversalis muscles.

The widening is when a large sac of long duration or a large recurrent sac has been pushing through. Wearing of trusses over a long period likewise causes definite atrophy of muscles and widening of the triangle. It is therefore impossible to do a repair by attempting to draw muscle over to the inguinal ligament without undue tension by the usual oft-repeated catgut methods of operation. Some type of living suture method must be used to build a new protective wall. A method of using strips of fascia from the rectus sheath as sutures to span this space and build the new wall was described and illustrated with lantern slides. An experience of ten years with huge primary and recurrent hernias was reported. Fascia is autoplastic, heals readily into contiguous structures and becomes permanent tendinous grafts. Previous publications with reported cases were cited as convincing evidence of the efficacy of this method. The method is important in economic rehabilitation, as it places people back at work who would otherwise be dependent on family or charity.

Dr. John F. Casey gave a brief talk on Peripheral Vascular Disease, particularly emphasizing the early symptoms and diagnostic points. He felt that early diagnosis and early treatment would result in the saving of many extremities. The Pa-va-ex machine was demonstrated at the close of the meeting.

Dr. Francis P. McCarthy, Pathologist at the hospital, gave a report of a group of unusual and interesting cases with gross and microscopical pathological specimens.

One unusual breast tumor, an adenomyxomafibrosarcoma was shown. This tumor was of twenty years' duration and began as an adenofibroma.

A case of lymphosarcoma involving the mediastinal and retroperitoneal lymph nodes with diffuse involvement of both kidneys was demonstrated. The kidneys were tremendously enlarged, each weighing over 700 grams and were the only organs involved other than the lymph nodes.

An embryonal sarcoma of the right kidney, in a child of two years, which involved a greater part of the abdominal cavity was also shown.

A specimen of lung showing typical silicotic nodules in an advanced case of silicosis was demonstrated together with the microscopical sections.

An enormous fatty cirrhosis of the liver weighing over 7000 grams and a large aneurysm of the ascending and transverse arch of the aorta were demonstrated.

An interesting specimen of an adenocarcinoma of the stomach and a rheumatic heart, weighing 1120 grams, from an eighteen-year-old boy who had rheumatic fever in early childhood, were demonstrated.

THE FAULKNER HOSPITAL CLINICAL MEETING

The regular monthly clinical meeting was held at The Faulkner Hospital on Thursday afternoon, March 5, at 5:00 P.M.

One of the cases which had come to autopsy during the month was that of a male, forty-eight years of age, who presented a typical picture of infection involving the larynx. Although diphtheria was suspected, the culture was negative. Examination of the larynx showed a congested and swollen epiglottis and vocal cords. It was not possible to see beyond the vocal cords. A short time after the patient arrived in the hospital he coughed up a perfect cast of the trachea and beginning of the bronchi. From this cast diphtheria organisms were obtained. Despite large doses of antitoxin, laryngeal obstruction developed and a tracheotomy was done. The patient succumbed from what appeared to be general

toxic reaction. At autopsy a typical picture of laryngeal diphtheria was present. Marked congestion and edema filled the alveolar spaces. From the lungs a culture of streptococcus viridans was obtained. In certain places in the lungs there was a hyaline-like membrane such as is found in cases of the epidemic disease generally called influenza, but the distribution of this membrane was different from that found in the epidemic disease. An interesting feature of the case was a leukocyte count of 42,400 with 92 per cent polynuclears, which is rather high for infection with diphtheria and would not be expected in a streptococcus viridans which was the only other organism found at the autopsy.

The other case was that of a woman seventy-eight years of age who had a terminal infection with high fever, gradually rising pulse rate and elevated respirations with a moderate leukocytosis. There were a few crackling rales at the base of one lung, and it was felt without doubt that the case was one of terminal pneumonia in an old woman. This case demonstrates the value of having a postmortem examination on all cases no matter how simple they seem, because there was no pneumonia. The cause of death was a staphylococcus aureus septicemia with marked involvement of the heart muscle. Microscopic abscesses were found elsewhere, especially one in the brain which was just about to burst through into the meninges and, had she survived a day or so longer, it would have undoubtedly produced a staphylococcus meningitis. Another interesting feature in the case was the practically complete occlusion of the coronary arteries without pronounced cardiac infarction.

Following the presentation of these two cases Dr. Richard C. Eley talked on the clinical applications of extracts obtained from the human placenta. He called attention to the fact that there were two extracts at the present time, one a 2 per cent salt extraction with the resulting protein which had the power of neutralizing the poison of poliomyelitis, preventing measles, blanching the scarlet fever rash and neutralizing diphtheria toxin. Although this extraction has these four qualities, the practical application of it at the present time has been in the prevention of measles. He called attention to the fact that attempts to prevent measles had been tried by the use of serum from patients convalescing from measles, adult human serum and adult human whole blood in addition to the use of this placental extract. The advantage of the placental extract is that it can be used in smaller quantities than either adult whole blood or adult serum. Since it is put out by the Commonwealth of Massachusetts ready for use, it is easier to obtain than the serum from a convalescent patient.

He then showed figures to demonstrate that this placental extract is more effective in preventing measles or making the disease milder than the serums or the whole blood. He called attention to the fact that if this placental extract is taken by mouth it will reverse the Dick and Schick tests temporarily. Just what clinical application can be made of this has not as yet been decided.

He then called attention to another extract of the placenta which has a coagulating action upon human blood. He emphasized the importance of realizing that this coagulating action was not present in the extract of the placenta which is distributed by the Commonwealth of Massachusetts. The extract of the placenta used for the prevention of measles is sterilized by passing through the Berkefeld filter. The coagulating agent in the placental extract will not go through the filter and therefore cannot be sterilized. It is given by mouth and one of the difficulties is the tendency of the gastric juices to destroy it. The results upon children who are hemophiliacs have been very remarkable in many cases. In some cases it has not been so successful. In adults the results have not been so satisfactory as in children, and it is felt that there is some point in regard to the destruction of the substance in the gastric juice which has not as yet been overcome. In addition to the value of this preparation in children with a tendency to bleed, it has been successful in stopping the oozing from raw surfaces after adenoid operations thus saving the packing of the nasopharynx, stopping the bleeding from cleft palate operations and also stopping hemorrhage from the pockets from which teeth have been extracted. Some interesting results have also been obtained in cases of prolonged menstruation. Up to the present time the results on cases of purpura and leukemia have not been encouraging.

This extract of the placenta which contains the coagulant can be given by mouth, intramuscularly or applied locally.

WILLIAM HARVEY SOCIETY

The William Harvey Society held its regular monthly meeting on February 14, 1936, with Dr. Elmer W. Barron presiding. Dr. L. Emmett Holt, Jr., Associate Professor of Pediatrics at Johns Hopkins University School of Medicine, spoke on the "Significance of the Fats in Infant Nutrition." High fat diets in infant feeding have been advocated periodically since Biblical times. A wave of enthusiasm for this type of feeding, which spread to England and this country, was started by Biedert in Germany in the latter part of the nineteenth century. In its trail came a reversal of feeling, fats being deprecated and avoided because of their supposed tendency to produce diarrhea, their demonstrated inhibitory action on the stomach, a belief that they were responsible for certain types of anemia and that their prolonged use in large quantity might lead to the syndrome of "fat intolerance". Particular opprobrium has been attached to the fat of cows' milk and, in the belief that fats are species specific, many attempts have been made to imitate the fat of human milk in artificial feeding. This doctrine has survived and finds expression in the current practice of feeding not more than 35 per cent of the calories as fat when cows' milk is used, and also in

the use of various fat mixtures designed to imitate breast milk fat.

Dr. Holt and his collaborators were led to question the soundness of these views. They undertook to reinvestigate the question of the species specificity of milk fat and to make comparative studies of various fats in infants from the point of view of ease of absorption, of their inhibitory influence on the stomach, their supposed hemolytic effects and their internal utilization.

Observations on human milk fat indicated there was comparatively little species specificity. The fat could be greatly altered by changes in the character of the fat of the diet; this had been previously reported by Engel whose work was generally overlooked.

The conditions influencing fat absorption in general were studied. It was found that the size of the fat particles is not a factor of importance; the quantity of protein and carbohydrate present exert no appreciable influence, but minerals, particularly calcium in excess, may impair fat absorption.

The retention of different fats by infants was studied. These were selected with a view to demonstrate the effects of varying the composition of the fat. It was found that the completeness of absorption could be predicted within narrow limits from a knowledge of the component fatty acids. The shorter chain fatty acids were more readily absorbed than those with longer chains. The presence of an unsaturated linkage in the fatty acid chain favored absorption. The evidence as to whether more than one double bond was advantageous was not conclusive, but there was suggestive evidence that this was the case. No evidence was obtained that the volatile fatty acids are irritating or cause diarrhea, as had been claimed. It was found that several vegetable fats, notably olive oil and soya bean oil, were even better absorbed than average human milk fat. The "imitations" of human milk fat were not so well absorbed as the human milk fat itself. They represented little or no improvement over butter and one preparation was less well absorbed than butter. The differences in absorption found with normal infants on these various fats were not so great as to be of practical importance. In a series of premature infants, however, who are notoriously poor fat absorbers, the substitution of a more digestible vegetable fat for butter caused a significant increase in absorption which was promptly reflected in the weight curve.

It has been known for many years that fats inhibit the motility and secretion of the stomach. The phenomenon is due to a humoral mechanism released after fat enters the duodenum. Lim and others have extracted a chalone from the duodenum which is capable of producing this effect, when introduced into the circulating blood. Cameron and Tidwell in Dr. Holt's laboratory made comparative studies of the inhibitory effect of different fats, and found that the fats which produced the greatest inhibition were those which were most readily assimilable. The purpose of this inhibitory phenomenon remains obscure, but at least it does not appear to be harmful.

In the past it has been maintained that fats gave rise to increased blood destruction, although recent work has cast doubt upon this. The problem was restudied in Baltimore with the collaboration of Dr. Josephs in a series of infants whose urobilin output was determined on various high fat diets. The response was uniform regardless of the type of fat used: the urobilin output rose promptly but within a few days it fell again, being maintained at a level almost twice as high as the normal for that individual. It did not at any time reach the high values found in infections or hemolytic anemias. The preliminary marked rise appeared to be due to a washing out of stores, since it could be produced by mineral oil alone. The protracted effect could not be produced with mineral oil. It is not proved that the sustained increase in urobilin output is due to blood destruction. Other evidence of blood destruction was not found, but by exclusion it would seem that a mild degree of increased blood destruction is responsible for the increased urobilin output.

Attempts were made to study the internal utilization of various fats in infants. Intravenous injections of various emulsified fats revealed no differences in the rate of removal from the blood. This may, however, have been due to the fact that in all instances egg lecithin was used as an emulsifying agent, giving the fat particles a uniform coating. Studies were also made by Nichols and Myers in Dr. Holt's laboratory on the alimentary lipemic curve after the ingestion of various fats. When the quantity of fat ingested for the test is sufficiently large, a striking alimentary lipemia is exhibited by the infant. If, however, the infant is maintained upon a high fat diet for some days or weeks, the lipemic curve induced by the same test dose of fat gradually diminishes, as the organism acquires an increased ability internally to dispose of fat. This same phenomenon has been observed in animals by Bang and by Leites. Whether it is due to a "training" of the tissues to utilize fat or to some circulating hormone elaborated in larger quantities upon demand is still unsettled. In an infant adapted to utilize a large quantity of one fat, other fats were substituted for the test meal in the hope of detecting by variations in the alimentary lipemic curve, whether this fat-adapted state was specific for particular fats. No such specificity was demonstrated.

NEW ENGLAND ROENTGEN RAY SOCIETY

The February meeting of the New England Roentgen Ray Society was held at the Massachusetts General Hospital on the evening of February 21, 1936. After the brief business of the Society was over, Dr. Holmes presented the various speakers of the evening.

Dr. A. O. Hampton spoke on "Boeck's Sarcoid." The etiology of this condition is unknown, it may involve any tissue of the body, but the lesions in the lungs and bones are of chief interest to the roentgenologist. Where the lungs are involved,

there is a symmetrical enlargement of the lung roots with or without a diffuse parenchymal lesion. There are no pulmonary symptoms; the condition is not malignant; and the lesions disappear of their own accord without treatment within six to eight months. One-third of the patients have bone changes which are typically in the fingers, causing fusiform swellings, and present x-ray lesions varying from pin-point destructive areas to large areas of pressure erosion. Biopsy of involved glands shows multiple tubercles without giant cells or central necrosis. Patients with erythema nodosum may present the same chest picture. In some cases, sarcoid presents itself only in the pulmonary manifestations. Dr. Hampton showed x-rays of several of these patients.

Dr. Frank Hunter spoke on "Spray Radiation in the Treatment of Polycythemia Vera and Erythroblastic Anemia." In both these conditions there is an overactivity of the bone marrow. Patients with polycythemia vera have pains in the bones, enlarged spleens and dusky complexions. Two cases treated by spray radiation were discussed and blood charts shown. About 1000 r units were given over a period of two to three months in each case. All the blood elements fell and the red count stayed normal for some years. Thus far, spray radiation seems to be the most successful treatment in this condition.

Erythroblastic anemia of Cooley occurs chiefly in infants and young children causing extreme pallor, weakness and an enlarged liver and spleen. These patients often have Mongolian facies and may have pathological bone fractures, and the blood picture presents numerous nucleated red blood cells. One such case has been treated by x-ray and the nucleated reds have fallen, while the red count has risen to a more nearly normal value.

Dr. J. M. Robinson spoke on "The Radiographic Demonstration of Rupture of the Intervertebral Disc." This condition may occur in the cervical and dorsal region, but the condition occurs more commonly in the lumbosacral region and gives signs of a cord tumor. The soft nucleus of the intervertebral disc may rupture posteriorly or into the vertebral bodies as well as anteriorly. Two-thirds of the cases give a clinical picture of low back pain, typical of sacro-iliac strain. Lantern slides were shown to demonstrate the use of lipiodol to show the rupture. In 50 per cent of these cases, there is no visible narrowing of the disc. An increase in the spinal fluid protein is a constant finding. Approximately 50 per cent give a history of trauma and 90 per cent of the group occurring in the lumbosacral region between the fourth and fifth lumbar, and fifth lumbar and first sacral vertebrae. In 95 per cent of the cases the pain disappears immediately following operation.

Dr. H. O. Peterson spoke on "Analysis of 100 Cases of Ureteral Stones." Only those cases in which the indisputable diagnosis of this condition was made clinically were selected. Twenty-one out of the 100 cases had given no x-ray evidence, but reëxamination of the plates found stones in 96 per cent of the cases. The x-ray tube should be centered so that the bones of the pelvis are not superimposed on the orifice of the ureter. Eighty per cent of all ureteral stones are found within the boundaries of the pelvis, and 66 per cent are located in the small area near the ureteral orifice. All the small stones are in this latter area.

Dr. R. Lingley spoke on "Malignant Small Bowel Tumors." He presented four cases of this condition. Dr. Lingley concludes that these malignant tumors of the small bowel may obstruct the lumen, but more often dilate it and there is usually a large, soft, tissue mass palpable.

The last paper was by Dr. R. H. Schatzki on "The Diagnosis of Tumors of the Colon." He pointed out several practical points in the examination of the colon. First, the patients should be prepared carefully. Secondly, the most important part of the examination is a careful fluoroscopic study in which every inch is carefully observed. The rectum should be slowly filled and the enema stopped following this because the peristalsis will fill the sigmoid and part of the colon without more barium. The less barium used, the clearer the outlines of the colon will be. Spot films are taken of any suspicious areas. The patient should be reëxamined by fluoroscopy after evacuation of the barium.

Obstruction is the most common finding, and several plates were shown to demonstrate the different kinds of obstruction. The differential diagnosis between stenosis due to diverticulitis and that due to tumor may be difficult. The diagnosis of small tumors was stressed and the typical findings in cases of polyps shown. Spasm is often present with polyps. These conditions are often only found in the post-evacuation studies.

HARVARD MEDICAL SOCIETY

The Harvard Medical Society met at the Peter Bent Brigham Hospital February 25, 1936, Dr. Elliott C. Cutler presiding. The medical case was presented by Dr. Charles B. Kimmel. An eighteen-year-old boy, a known diabetic for nine years, had been referred to the hospital for medical observation and treatment of an abscess of the left thigh. He had been seen on two previous occasions because of threatened diabetic coma, which had been precipitated on both occasions by upper respiratory infections. He had been well until six days before the present admission, when he developed a localized abscess in the left thigh at the site of a hypodermic injection of insulin. Physical examination was negative except for the abscess, which was subsequently incised and drained. He was placed on the usual diabetic régime, but was given protamine insulinate, which decreased his insulin requirements, at 10 p.m. and 7 a.m. Dr. Marshall N. Fulton remarked that this was the second case which had been given protamine insulinate at the Brigham Hospital. It was admin-

istered at 5 p.m. and somewhat decreased the patient's tendency toward high blood sugar values in the morning. Dr. Elliott C. Cutler commented on the obvious immaturity of the boy and emphasized the fact that many patients with diabetes show associated endocrinological abnormalities.

Dr. Richard C. Durant presented the surgical case. A forty-year-old woman entered the Brigham Hospital on Oct. 17, 1935, complaining of abdominal pain and vomiting of twenty hours' duration. Her distress had begun as nausea, and then pain had appeared in the right upper quadrant which soon became of a colicky nature. She had had a small, soft, apparently normal bowel movement after the onset of her pain. Her local physician reported that examination of her abdomen was negative, and that 15 milligrams of morphine were required to relieve her pain. Some ten hours after the onset of the pain she vomited material containing blood. The next day she entered the Brigham Hospital. Examination on entry revealed abdominal spasm and tenderness, most marked on the left side. Rectal and pelvic examinations were negative. The white count was 12,200, 92 per cent polymorphonuclear leucocytes; the red count was five million, and the hemoglobin was 85 per cent. Exploratory abdominal laparotomy was performed, and revealed a serosanguineous exudate in the peritoneal cavity, and numerous small necrotic areas scattered over the peritoneum. The bowel was hemorrhagic in appearance, and the pancreas was found to be several times its normal size. The pancreatic capsule was incised, and the abdomen was closed with drainage. She experienced a moderate amount of postoperative nausea, vomiting and pain, and had several recurrent attacks of these symptoms. A draining sinus persisted, with the discharge of approximately 300 cubic centimeters of fluid daily. On December 22, 1935 after a severe coughing spell a piece of necrotic tissue was found in the dressing. Microscopic study did not reveal the nature of this tissue, because of its poor condition, but it was assumed to be pancreatic tissue. Following the discharge of this tissue the amount of drainage steadily decreased. Her temperature, which had been persistently elevated to 101° to 102° Fahrenheit, subsided to 99°. A fluoroscopic study of the gastrointestinal tract revealed a hyperirritable stomach, with large rugae, and a five per cent residue. There were numerous shallow defects on the lesser curvature which were interpreted as representing ulcerations. The duodenum emptied satisfactorily, but was irregular in outline, due to adhesions. Dr. Cutler remarked that inasmuch as a large portion of this patient's pancreas had been destroyed, it would be reasonable to expect a variation in sugar tolerance which might simulate diabetes. Although her sugar tolerance was normal shortly after her operation it subsequently became very similar to that of a diabetic patient. Dr. Russell Wilder commented on the fact that, even though large portions of the pancreas were often destroyed by pancreatitis, only rarely do such cases develop diabetes. This fact illustrates the large margin of reserve function possessed by this organ.

Dr. Russell Wilder, chief of the Department of Medicine of the Mayo Foundation, delivered the address of the evening. His topic was "Spontaneous Hyperglycemia." The clinical condition designated by this term might better be known as "paroxysmal hypoglycemia", because the syndrome is characterized by periodically recurring attacks or fits. This condition was recognized clinically in 1927, when a patient was observed at the Mayo Clinic suffering from symptoms which were similar to those of insulin shock, and were immediately relieved by the administration of glucose. The blood sugar during an attack was found to be only 30 mg. per cent. Operation revealed carcinoma of the pancreas with metastases. Postmortem studies demonstrated that the carcinoma was composed of cells which resembled morphologically the cells of the islands of Langerhans. Extracts of the metastases in the liver when injected into rabbits produced variations in blood sugar levels identical with those produced by insulin, confirming the belief that this metastatic tissue was functionally active. Subsequent cases which have been seen at operation have been either adenomas of the islets, or very early carcinoma without metastases. Although extracts of such tissues have been shown to have activity like insulin it cannot be said with absolute certainty that such tissue is hypersecretory. However the clinical evidence points toward this belief. Very recently a second case with metastatic involvement of the liver has been seen at the Mayo Clinic, and extracts of the metastases have given results similar to those obtained in the first case described.

Some cases of hypoglycemia have been encountered in which no islet tumor could be demonstrated, either at operation or at postmortem study. Some of these cases have been found to have abnormalities in the anterior hypophysis, e.g., a chromophobe adenoma. Cushing first described low blood sugar levels in patients with pituitary lesions. Many of these latter cases suffer symptoms from their hypoglycemia, although an occasional patient may have a persistent low blood sugar level without discomfort. It is believed that the anterior lobe of the hypophysis elaborates a hormone which is antagonistic to insulin in its action. The exact mechanism of this action is at present unknown, although it is believed by Long that the pituitary mediates its influence through the adrenal cortex.

It has been suggested that disturbed function of the adrenal cortex may be responsible for some cases of hypoglycemia. Clinical observations have not supported this hypothesis. In 150 cases of Addison's disease observed at the Mayo Clinic marked hypoglycemia was never observed.

It seems unlikely that the thyroid dysfunction is of importance in causing severe hypoglycemia, since this symptom is not observed following subtotal or total thyroidectomy.

Experimental and clinical observations have indicated that the presence of adequate hepatic func-

tion is essential for the maintenance of normal blood sugar levels. The reserve power of the liver in this respect is enormous; at least 80 per cent of the hepatic tissue must be removed in order to cause a lowering in the blood sugar. Complete removal of the liver brings about marked symptoms of hypoglycemia, which are quickly alleviated by the administration of glucose. Severe liver damage such as is encountered in advanced cirrhosis, or extreme fatty degeneration results in low blood sugar values.

Paroxysmal hypoglycemia may be precipitated in perfectly normal men by sufficiently strenuous exercise (e.g., a marathon race). Some individuals apparently are less well able to manufacture blood sugar than others and less severe exertion causes hypoglycemia in them.

Hypoglycemia has been observed to occur in newborn infants of diabetic mothers. Postmortem study has revealed very large pancreatic islets in some, but not all, of these infants. It is supposed that the pancreas of the fetus functions more actively than in adults in order to supply insulin for the maternal organism, and that this hyperfunction continues after birth and results in a true hyperinsulinism. Frequent oral feedings with glucose immediately after birth, supplemented with subcutaneous administration of 10 per cent glucose solution, have prevented the loss of infants from hypoglycemic death. Dr. Wilder suggested the possibility that some of the convulsions seen in children of nondiabetic mothers within the first few days after birth might be due to unrecognized hyperglycemia, instead of the usually diagnosed "birth injuries."

The treatment of hypoglycemia in adults entails the administration of glucose either orally or intravenously in the acute stages. Mild cases should be prescribed small, frequent meals of the same general type as that recommended by diabetic patients in order to avoid stimulating the overproduction of insulin. Exercise should be curtailed, and the patient warned to be on his guard for early manifestations of a hypoglycemic reaction. Severe cases frequently are due to islet tumors, which occasionally are found to be malignant. Operation should be advised in all severe cases. In cases in which no tumor has been found at operation some surgeons have performed a "subtotal resection" of the pancreas with good results. Recently in one such case a silk ligature was carried around the body of the pancreas and ligated anteriorly to the divisions of the celiac axis and tied snugly. The patient developed mild diabetes and previous attacks of hypoglycemia ceased.

CLINICAL MEETING OF THE FIFTH SURGICAL SERVICE AND SURGICAL RESEARCH LABORATORY OF THE BOSTON CITY HOSPITAL

A clinical meeting of the Fifth Surgical Service and Surgical Research Laboratory of the Boston City Hospital was held in the Cheever Amphitheatre on March 25, 1936, Dr. Irving J. Walker presiding. The first paper of the evening was presented by Dr. Stanley J. G. Novak, who spoke on "Cross Circulation Studies in Surgical Shock." There are four main theories concerning the etiology of surgical shock, which may be briefly summarized: (1) That of Crile, who believes that there is a paralysis of the vasomotor center. (2) That of Cannon, who believes that as a result of trauma and tissue destruction there is liberation of some sort of toxic substance, which is absorbed into the blood stream and produces shock. (3) That of Blalock, who believes that shock is produced by hemorrhage, merely as a result of lowered blood volume. (4) That of Freeman, who attributes the condition to overactivity of the sympathetic system. The latter worker observed the onset of shock (as indicated by lowering of blood volume), after the prolonged intravenous injection of physiological quantities of adrenalin, and after prolonged and profound hyperactivity of the sympathetic nervous system in animals showing "sham rage" (hypothalamic preparations).

Dr. Novak accepted the fall in blood pressure as the criterion of shock in his animals, and used the cross circulation method of Delezenne, in which the hind limb of dog "B" is isolated from its body except for the nerve supply, and all blood for the limb is supplied by means of cross circulation from dog "A". This isolated limb was subjected to extreme trauma, and by following the blood pressures in the two dogs by means of arterial cannulae, it was determined which animal went into shock. If shock were due to absorption of toxic substances, or to blood loss, animal "A" would be expected to show marked drop in blood pressure, while if shock were due to pain and overactivity of the sympathetic nervous system, dog "B" would be expected to exhibit the lowered blood pressure. The amount of hemorrhage into the limb, and total blood loss were accurately determined after the completion of the experiment by weighing the limb, and by determining the amount of hemoglobin present on the sponges and instruments.

In a long series of experiments it was consistently observed that dog "B" sustained its normal blood pressure, and showed no evidence of shock, while dog "A", the "perfuser" dog, went into shock with marked fall in blood pressure.

Calculations of the loss of blood in the leg and on sponges showed that there was an average loss of approximately 23 per cent of the total blood volume. It was found experimentally that a hemorrhage with the loss of 20 per cent of the blood volume was sufficient to cause shock.

By crushing the other leg of dog "B", which had previously been denervated, and observing the vasomotor responses of the isolated leg, it was found that dog "B" went into shock. The isolated leg showed vasoconstriction, and although dog "A" showed variations in blood pressure, there was no shock.

Retransfusion of the dog exhibiting shock promptly elevated the blood pressure, and alleviated the symptoms of shock.

These studies of Dr. Novak confirm the theory of Blalock that "shock is hemorrhage, and hemorrhage is shock".

Dr. Charles C. Lund spoke on the subject "Arterial Embolism", stating that emboli in the larger arteries must arise from either the pulmonary vein, the left side of the heart, the aorta, or some portion of the artery above the site of the embolus. In extremely rare instances an embolus may pass through a patent foramen ovale, although there is little tendency for blood or emboli to pass through such an orifice, even though existent, since the blood pressures in the two sides of the heart are practically equal under normal conditions. If, however, there is an embolus to the pulmonary artery in a person with a patent foramen ovale, there is an elevation of the blood pressure in the right heart, and a second embolus may be forced through the opening into the left auricle.

In general there are two types of emboli, the first and most usual being a hard plaque of material which occludes the vessel at one point only, and the second being a soft "mushy" clot, which may fill up the whole of the artery peripheral to the occlusion. Only the first type is amenable to treatment by embolectomy.

In former years many more patients were subjected to the operation of embolectomy than at the present time. The use of the suction pump on the extremity, peripheral to the embolism, has proved successful in establishing collateral circulation in many instances. A recent report of the use of papaverine hydrochloride in patients suffering from emboli has suggested that this drug may be of value in relieving the arterial spasm which occurs following the occlusion of a vessel with an embolus.

Patients experiencing peripheral emboli are extremely poor surgical risks, and even though fifty per cent of those operated upon within twelve hours after the onset of embolism get complete restoration of circulation, many will die in the hospital of some other cause. A survey of cases subjected to surgical treatment of emboli has shown that 25 per cent leave the hospital with good circulation in the affected limb; 25 per cent leave the hospital after amputation of the limb; and 50 per cent die while in the hospital. The mortality of such patients is 90 per cent within the first two years following the embolus.

Dr. Novak stated that papaverine had been used at the Peter Bent Brigham Hospital in the treatment of emboli with very discouraging results.

Dr. E. Everett O'Neil presented a paper on "Gangrene and Impending Gangrene of the Extremities: Considerations and Treatment." He emphasized the importance of careful instruction of patients with vascular disease as to the proper care of the feet. In appraising the true state of the circulation in an extremity it is important to differentiate the true occlusive type and the spastic type. The index of arterial sufficiency, the histamine test, skin thermometry before and after spinal anesthesia, and the Landis test are all of distinct value in making this appraisal.

Application of the suction apparatus to the affected extremity is often effective in elevating the skin temperature, and alleviating pain, and may permanently aid cases with frozen feet, or arterial emboli. Dr. O'Neil does not believe that cases of Buerger's disease should be so treated, however.

Cases of diabetic gangrene should be treated as surgical emergencies, and after amputation the stump should be treated with primary closure. The guillotine amputation is of distinct value in many such cases, and should be more widely used than it is at present.

"Renal Colic in the Male Caused by Vesiculitis" was discussed by Dr. Augustus Riley. The anatomical location of the seminal vesicles on the posterior surface of the bladder and overlying the lower portion of the ureters is such that swelling may occlude the ureteral lumen and cause back pressure. Such occurrences have been observed in a considerable number of cases by Dr. Riley, and these cases were found to have elevated temperature, abdominal spasticity, and pain fully as severe as that caused by the passage of a ureteral stone. The only method of establishing this diagnosis is by means of cystoscopy, and pyelograms. Cystoscopically a characteristic swelling and edema are observed in the trigone at the ureteral orifice, and the ureter can be catheterized for a distance of about two centimeters only. Dilatation of the ureter often will result in drainage of infected urine which may contain red blood cells. Such blockage may occasionally be the underlying factor in the production of pyelitis.

Chronic vesiculitis may cause pain localized to the inguinal regions, which is often mistakenly diagnosed as appendicitis or "gas pains".

Dr. Irving J. Walker and Dr. W. B. Castle presented a case, and discussed the "Dietetic Problems Associated with Total Gastrectomy." The patient was a forty-one year old male who was first seen one and one-half years previously, complaining of loss of appetite, general malaise, and weakness of three months' duration. Except for loss of appetite there were no symptoms of gastrointestinal disease. The past history was negative. X-ray studies showed a "fixed stomach" with poor peristaltic activity, and the diagnosis of gastric carcinoma or gastric ulcer with gastritis was made. The physical examination was negative, and laboratory studies, except for a low gastric acidity, were within normal limits. Dr. Walker performed a total gastrectomy, making an end-to-side anastomosis between the esophagus and jejunum, and an enteroenterostomy between the jejunal loops. A Levine tube was put in place at the time of the operation, and brought into the loop of jejunum proximal to the stoma. This was left in place for four days postoperatively. The abdomen was drained under the left costal margin to remove what serum might accumulate. The patient was kept in an oxygen tent for three days after the operation, and fluids were given by hypodermoclysis

and intravenously up to 3000 cc. daily. A soft-solid diet was given after the third day.

Gross examination of the stomach after removal showed what appeared to be an ulcer two centimeters in diameter. Microscopic studies showed a carcinoma simplex with numerous mitoses. No metastases were found. Dr. Walker believes that patients with histories and x-ray findings such as were observed in this case should be considered as having carcinoma until proved otherwise.

Dr. Castle stated that loss of weight is not to be expected in cases of carcinoma unless the disease is widespread, or involves the essential organs of digestion. This patient lost thirty pounds after his operation, and had weighed only 100 pounds for several months. This weight loss might be due to metastatic disease, but there was no indication of such invasion. It could not be attributed to the loss of the chemical products of the stomach. His basal metabolic rate was normal, and there was no increase in the fat in the stools. Although he showed a very great alimentary hyperglycemia and glycosuria, the loss of calories in this fashion was insignificant compared with the total caloric requirements of his body, with his dietary intake. The true explanation of his failure to gain weight was finally found to be his refusal to take in adequate amounts of food. By using concentrated foods, and much persuasion, he had been induced to take a 2000 caloric diet for one week, with the result that he gained three pounds. The whole problem in this case was the effort to overcome the patient's reluctance to eat.

This case was also interesting since it illustrated the fact that pernicious anemia does not occur after total gastrectomy, although it is known that the stomach is of importance in preparing the antianemic principle. This patient did not develop pernicious anemia because of the fact that there is a store of "liver extract" in the liver, sufficient to last for many months, because of the fact that liver extract can be absorbed from food, and because the pyloric glands have a physiological analogue in Brunner's glands of the duodenum, which act as a source for the "intrinsic factor". Because of the latter two reasons there is no reason to suppose that gastrectomy patients would develop pernicious anemia. It is true that several cases of pernicious anemia have been reported following gastrectomy, but it is believed that these patients developed the disease as a result of inadequate diets, and not directly because of the loss of the stomach. In true pernicious anemia there is probably a degeneration of the whole gastrointestinal tract, with many abnormalities such as poor absorption, and inadequate formation of the intrinsic factor.

Dr. David D. Berlin reported on the "End Results of Total Thyroidectomy for Chronic Intractable Heart Disease." He pointed out that cases subjected to this form of therapy must be very carefully selected. Surgical treatment has no place in heart disease if any benefit can be obtained from medical treatment, if the basal metabolic rate is low, or if the disease is rapidly progressing. One hundred and eighty-five cases were analyzed, representing the results in twenty-six clinics. The average operative mortality in the various clinics was between five and eight per cent. Since the adoption of local anesthesia, and careful selection of cases, there have been no operative deaths at the Beth Israel Hospital. A summary of the results in the two groups of cases was presented.

Results	Beth Israel Hospital	Other Clinics
A. Congestive Failure		
Excellent	31%	31%
Moderate improvement	17%	32%
Slight improvement	21%	18%
No improvement	31%	18%
B. Angina Pectoris		
Excellent	35%	53%
Moderate improvement	26%	33%
Slight improvement	16%	9%
No improvement	23%	5%

In summary, Dr. Berlin stated that 132 of the 185 patients subjected to the operation received benefit from the procedure. He believes that it is a valuable method of treatment in properly selected cases.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Annual Meeting of the Essex North District Medical Society will be held Wednesday, May 6, 1936, at the Hotel Hawthorne, Salem, Mass.

PROGRAM

12:30—Dinner.

2:00—Business meeting—Nomination of officers, adoption of changed By-Laws.

3:00—Fundamentals in Cancer Treatment—Grantley W. Taylor, M.D., Harvard Medical School, Boston.

The Rôle of the Pathologist in the Diagnosis of Neoplasms—Béla Halpert, M.D., Head of the Division of Pathology, The Jewish Hospital, Brooklyn.

Censors' meeting at the Hotel Bartlett, Haverhill, Thursday, May 7, 1936, at 4:00 P.M.

E. S. BAGNALL, M.D., *Secretary-Treasurer.*

THE WACHUSETT MEDICAL IMPROVEMENT SOCIETY

Under the auspices of this Society a Medical Historical Pageant will be presented at the Rutland State Sanatorium on April 30, 1936, at 8:00 P.M.

PROGRAM

The Story of Early Medicine in Massachusetts given by students of Tufts College Medical School.

Director—Professor B. Spector.

Colonial Period (1620-1700)

Indian Medicine Man (1620-1640). R. A. Johnson, '37.

Deacon Dr. Samuel Fuller (1580-1633). M. V. MacKenzie, '38.

Gov. John Winthrop, Mass. (1588-1649). W. E. Greer, '36.

Gov. John Winthrop, Jr., Conn. (1606-1676). R. K. Talkov, '37.

Rev. Thomas Thacher (1620-1678). D. Lichtman, '37.

Pre-Revolutionary Period (1700-1775)

Smallpox Inoculation:

Dr. Zabdiel Boylston (1679-1766). J. E. Burke, '38.

Rev. Cotton Mather (1663-1728). M. S. Allan, '38.

Dr. William Douglass (1691-1752). C. Djerf, '37.

Midwifery and Obstetrics:

Mrs. Samuel Fuller (fl. 1623-1664). Mrs. E. Lewis, '37.

Dr. James Lloyd (1728-1810). D. H. Gersh, '37.

Apprenticeship in Medicine:

Mr. Joseph Hartshorne (fl. 1752-1756). E. A. Guralnick, '37.

Dr. Silvester Gardiner (1708-1786). R. Femino, '37.

Mrs. Silvester Gardiner. M. I. Daly, '38.

Post-Revolutionary Period (1775-1894)

Cowpox Vaccination:

Dr. Benjamin Waterhouse (1754-1846). F. W. Lewis, '37.

Dr. Amos Holbrook, Milton Experiment (1753-1842). R. H. Fuller, '38.

First Ether Demonstration:

Dr. John Collins Warren (1778-1856). J. F. Conlin, '38.

Dr. Henry J. Bigelow (1816-1890). A. D. Ward, '38.

Dr. William T. G. Morton (1819-1868). L. I. Stellar, '38.

Dr. Oliver Wendell Holmes (1809-1894). O. B. Tibbetts, '36.

Mr. James Ballard, Director of the Boston Medical Library, will present an exhibition of Medical Books in connection with the Pageant.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, April 28, at 8:15 P.M.

PROGRAM

Presentation of Cases.

The Pathology of Vitamin Deficiencies. By Dr. S. Burt Wolbach.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held Monday, April 27, at 8:15 P.M., at the Boston Dispensary, 25 Bennet Street. Program: 1. The Significance of a Prominent S-Wave in Lead II of the Human Electrocardiogram. Dr. George Ravit. 2. Observations on Shock and Collapse. Dr. S. J. Thannhauser. 3. Unexplained High Basal Metabolism Rates in Heart Disease Without Failure. Dr. Heinz Magendantz. 4. Clinical Observations on the Treatment of Angina Pectoris. Dr. Joseph H. Pratt. 5. Some Effects of Diet Restriction on the Circulation in Patients with Heart Disease. Dr. Samuel H. Proger.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, APRIL 27, 1936

Monday, April 27—

*8:15 P.M. New England Heart Association at the Boston Dispensary, 25 Bennet Street, Boston.

Tuesday, April 28—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Pediatric Case Presentation. Dr. Francis McDonald.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

*8:15 P.M. Harvard Medical Society. Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance).

Wednesday, April 29—

Physicians' Art Exhibition. Doll & Richards Galleries, 138 Newbury Street, Boston.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 M. Clinico-Pathological Conference. Children's Hospital.

*4 P.M. Tufts College Medical School Annual Alumni Address. 416 Huntington Avenue, Boston.

Thursday, April 30—

Physicians' Art Exhibition. Doll & Richards Galleries, 138 Newbury Street, Boston.

*8:30-9:30 A.M. Clinic, Surgical and Orthopedic Staffs of the Children's Hospital, at the Children's Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Some Practical Aspects of Tuberculosis. Dr. C. H. Whitehurst.

12:30 P.M. Annual Meeting of the Massachusetts Society for Social Hygiene. Hotel Vendome (Empire Room), Boston.

*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.

*4:45 P.M. Henry Jackson Lecture Offered by the New England Heart Association. Boston Medical Library (John Ware Hall), 8 Fenway, Boston.

Friday, May 1—

Physicians' Art Exhibition. Doll & Richards Galleries, 138 Newbury Street, Boston.

*4:45 P.M. Henry Jackson Lecture Offered by the New England Heart Association. Boston Medical Library (John Ware Hall), 8 Fenway, Boston.

Saturday, May 2—

Physicians' Art Exhibition. Doll & Richards Galleries, 138 Newbury Street, Boston.

*10-12 A.M. Staff Rounds at the Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

April 23—Clover Hill Hospital. See page 803, issue of April 16.

April 23-24—Massachusetts Safety Conference. Hotel Statler, Boston.

April 27—New England Heart Association. See notice elsewhere on this page.

April 27—Springfield Medical Association, 8:30 P.M. at the rooms of the Springfield Academy of Medicine, 20

Maple Street. The Development of Non-Surgical Specialties. Dr. Allen S. Johnson. The Development of Surgical Specialties. Dr. Eugene W. Beauchamp.

April 28—Harvard Medical Society. See page 864.

April 29—Tufts College Medical School Annual Alumni Address. 4 P.M., 416 Huntington Avenue, Boston. Preventive Medicine and Public Health. Dr. William B. Keeler.

April 29 - May 9—Physicians' Art Exhibition. Doll & Richards Galleries, 138 Newbury Street, Boston.

April 30—Medical Clinic, Peter Bent Brigham Hospital. See page 855.

April 30—Massachusetts Society for Social Hygiene will meet at the Hotel Vendome (Empire Room), Boston, at 12:30 P.M.

April 30—Wachusett Medical Improvement Society. Medical Historical Pageant at the Rutland State Sanatorium at 8 P.M. See page 863 for complete program.

April 30 - May 1—The Henry Jackson Lectures. See page 855.

May 1, 2, 3 and 4—The American Association on Mental Deficiency. See page 610, issue of March 19.

May 11—American Medical Golfers Play in Kansas City. See page 710, issue of April 2.

May 11—American Association for the Study and Control of Rheumatic Diseases. See page 811, issue of April 16.

May 11-12—Annual Joint Meeting of the American Association of Medical Milk Commissions and Certified Milk Producers. See page 852.

May 12—American Heart Association, Inc. See page 712, issue of April 2.

May 12-16—The International Congress of Physical Medicine. See page 443, issue of February 27.

May 18—The American Neisserian Medical Society. See page 811, issue of April 16.

May 31 - June 1—International Cardiological Meeting, Royat (Auvergne) Assembly of Physiologists, Pathologists and Therapeutists. See page 754, issue of April 9.

June 15-19—The Executive Board of the Catholic Hospital Association will meet at the Fifth Regiment Armory, Baltimore, Md.

June 16-July 28—Summer Course in Bacteriology. See page 385, issue of February 20.

September, 1936—First International Conference on Fever Therapy. See page 1325, issue of December 26, 1935.

September, 1936—First International Congress of Sanatoria and Private Nursing Homes. See page 803, issue of April 16.

September 7-10—International Union against Tuberculosis. See page 554, issue of March 12.

October 19-23—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

May 6—Annual Meeting.

May 7—Censors' Meeting. See page 863.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

May 7—Thursday. Censors' Meeting.

May 13—Wednesday. Annual Meeting. Salem Country Club. Dinner at 7 P.M. Speaker: Dr. Paul White. Subject to be announced later.

R. E. STONE, M.D., Secretary.
88 Lothrop Boulevard, Beverly.

FRANKLIN DISTRICT MEDICAL SOCIETY

May 12—Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

May 6—Bear Hill Golf Club, Stoneham, at 12:15 P.M.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

May—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D., Secretary.
1236 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

May 21—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.
167 Newbury Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

April 29—Boston Medical Library, 8 Fenway, Boston. 8:15 P.M.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

May 7—Censors' Meeting. See page 713, issue of April 2.

May 13—Wednesday afternoon and evening. Annual Meeting of Society. (Time, place and details of program to be announced.)

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

Diagnostic Roentgenology. Edited by Ross Golden. 867 pp. New York and Edinburgh: Thomas Nelson and Sons.

Delafield and Prudden's Text-Book of Pathology. Revised by Francis Carter Wood. 1406 pp. Sixteenth Edition. Baltimore: William Wood & Company. $10.00.

Urethrography. Folke Knutsson. 150 pp. Stockholm: P. A. Norstedt & Söner. Swed. cr. 12:—net.

Das Ventrikulogramm. I. Teil, Röntgentechnik. Erik Lysholm. 74 pp. Stockholm: P. A. Norstedt & Söner. Swed. cr. 10:—net.

A Textbook of Roentgenology. The Roentgen Ray in Diagnosis and Treatment. Bede J. Michael Harrison. 826 pp. Baltimore: William Wood & Company. $10.00.

A Textbook of Obstetrics. For Students and Practitioners. Frederick C. Irving. 558 pp. New York: Macmillan Company. $6.00.

Adult Intelligence. A Psychological Study of Test Performances. Theodore Weisenburg, Anne Roe, and Katharine E. McBride. 155 pp. New York: The Commonwealth Fund. $1.40.

Studies from The Rockefeller Institute for Medical Research. Reprints. Volume 96. 637 pp. New York: The Rockefeller Institute for Medical Research.

Dental Infection and Systemic Disease. Russell L. Haden. Second Edition, Revised. 183 pp. Philadelphia: Lea & Febiger.

Abortion. Spontaneous and Induced. Medical and Social Aspects. Frederick J. Taussig. 536 pp. St. Louis: The C. V. Mosby Company. $7.50.

Synopsis of Clinical Laboratory Methods. W. E. Bray. 324 pp. St. Louis: The C. V. Mosby Company. $3.75.

Doctor Morath. Max René Hesse. 414 pp. Boston: Houghton Mifflin Company. $2.50.

A Guide to Psychiatric Nursing. F. A. Carmichael and John Chapman. Second Edition, Thoroughly Revised. 175 pp. Philadelphia: Lea & Febiger. $2.25.

BOOK REVIEWS

Lobar Pneumonia and Serum Therapy: With Special Reference to the Massachusetts Pneumonia Study. Frederick T. Lord and Roderick Heffron. 91 pp. New York: The Commonwealth Fund. $1.00.

This small handbook concerns the diagnosis and serum treatment of lobar pneumonia. A large proportion of the text is based on information gained through the Massachusetts Pneumonia Study from 1931 through 1935, which was financed by the Commonwealth Fund and carried out by the Massachusetts Department of Public Health.

Following introductory chapters dealing with the more general aspects of the problem is one concerning diagnosis and the proper selection of cases. In others, the methods of determining the type of pneumococcus and the properties of antipneumococcic serum are briefly considered. Subsequent chapters deal with the necessary precautions prior to serum administration, rules for the injection of serum and a description of serum reactions and a consideration of their treatment. Finally, the results of serum therapy are given.

The subject matter is presented simply and concisely. For those interested in more detailed information adequate references are given. Marginal notes emphasize most clearly the more important points.

Both authors are admirably qualified to write a book of this sort and it should prove of incalculable value to practicing physicians, particularly those with remote connections with the larger medical centers.

Obstetrical Practice. Alfred C. Beck. 702 pp. Baltimore: The Williams & Wilkins Company. $7.00.

The author's preface begins with this statement: "The purpose of this book is to present the essentials of obstetric practice to undergraduate students and young practitioners as concisely as is consistent with the requirements of a textbook"; and admirably has the author fulfilled his purpose.

There are thirty-nine chapters of text, beginning with a description of the ovarian cycle, and ending with instruction in the various standard methods of resuscitation of the newborn. Between these two is included a succinct presentation of the essential accepted facts of obstetrical practice. There is here no detailed discussion of physiological theories, but there is a clear factual recital of the whole subject. One who reads this book carefully will become acquainted with the whole range of obstetrics; he will even become familiar with the mechanics of labor and delivery which are described in enlightening detail; but he will not thereby equip himself as an expert obstetrician. The toxemias of pregnancy including hyperemesis and nephritis are discussed in fifteen pages. One page is given to pulmonary tuberculosis; almost a page, to both kinds of anemia. Heart disease is considered in little more than one page. To engross such subjects in such limited space the author has applied himself to the specific purpose of presenting to "students and young practitioners" the essentials as they are commonly accepted at present. Theories, divergent opinions and varied methods of treatment are scrupulously elided. The result is a clear didactic unit which must not be expected to serve as a complete exposition of the subject in hand.

The book is lavishly illustrated with excellent line drawings, well chosen and well placed. They illuminate the already lucid text. The paper is substantial, the print is large, the spacing is generous: a book, for the student or young practitioner, comfortable to read and profitable to study.

Lilly Research Laboratories, Dedication. Indianapolis: Eli Lilly & Company.

No one can read the account of the dedication of the Lilly Research Laboratories in this attractive book without a thrill of joy and pride that an American Pharmaceutical concern is so progressive. The building itself, 220 feet by 53 feet, is admirably arranged and houses much of the purely research work of the Eli Lilly Staff. To go through it without envy was more than one could expect of hard-working professors and doctors who gathered there on October 11 and 12, 1934, from various medical schools in the United States.

To the opening came more than a thousand guests of whom one-fourth were beyond Indiana state lines. In this dedicatory volume one can read the welcoming address of the chairman, Mr. Eli Lilly, the remarks of Mr. J. K. Lilly, the founder, fifty years ago of the first Lilly Laboratory, upon Research in Manufacturing Pharmacy, the conceptions of Dr. Irving Langmuir, regarding the Unpredictable Results of Research, Sir Frederick Banting's Early Story of Insulin, and Sir Henry Dale's paper on Chemical Ideas in Medicine and Biology. It is fortunate that the words of these famous men have been preserved and made available to all.

At the banquet, in the evening, given the visiting scientists and clinicians, there were brief after-dinner speeches by Mr. J. K. Lilly, Sir Henry Dale, Dr. Elliott P. Joslin, Dr. George R. Minot, Dr. Frank R. Lilly, Dr. George H. Whipple, Dr. Carl Voigtlin, and the Director of the Laboratories, Professor George H. A. Clowes. The volume also contains informal talks upon special problems of research which were given on the following day, together with a general description of the Lilly Research Laboratories.

Our congratulations, Eli Lilly Company, for the forward step you have taken to help humanity and the medical profession, and especially for the high ethical standards which now as always have characterized the endeavors of your organization.